EARLY ENTERAL FEEDING AND DELAYED ENTERAL FEEDING - A COMPARATIVE STUDY

Dissertation submitted to The Tamilnadu Dr.M.G.R.Medical University, in partial fulfillment of the requirement for the degree of M.S. (Branch-I General Surgery)

April 2013

ENDORSEMENT BY HEAD OF THE DEPARTMENT AND HEAD OF THE INSTITUTION

Certified that the dissertation entitled ***"Early Enteral feeding and Delayed Enteral Feeding – A comparative study"*** is a bonafide work done by the candidate **Dr. Barath Raj Kumar. S**, carried under my supervision. Certified further that to the best of my knowledge the work reported herein does not form part of any other thesis or dissertation on the basis of which a degree or award was conferred on an earlier occasion on this or any other candidate.

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DECLARATION FORM

I, Dr. Barath Raj Kumar.S, hereby declare that, I had carried out this work on *"**Early Enteral feeding and Delayed Enteral Feeing – A comparative study**"* at the Department of General Surgery, Kilpauk Medical College, during the period of July 2012 to November 2012. I also declare that neither this bonafide work, nor a part of it was submitted by me or others for any award, degree or diploma to any other University / Board either in India or abroad.

This is submitted to the Tamil Nadu Dr. M.G.R medical university, in partial fulfillment of the rules and regulations for the M.S. (Branch I - General Surgery) degree examination to be held in April 2013.

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Abstract

Aim:

To evaluate if early commencement of enteral nutrition compared to traditional management (delayed enteral feeding) is associated with fewer complications and improved outcome:

- In patients undergoing elective **/ emergency gastrointestinal surgery**;
- In patients with **acute pancreatitis.**

Design:

The following study was conducted in Kilpauk medical college and hospital. It is a prospective cohort interventional study, the source of the study being patients admitted in general surgery and surgical gastroenterology wards for either gastrointestinal surgeries or acute pancreatitis. The period of longitudinal observation was from July 2012 to November 2012. Inclusion and exclusion criteria were drawn up and only those patients satisfying both these criteria were included in the study. Patients admitted in my unit for GIT surgeries or acute pancreatitis constituted the test group while patients, while patients admitted in other units for similar disease processes constituted the control group. The sample size of the study was fixed at 100, the breakdown of which is as follows:

- **Test group (TG) – Patients were pooled from my unit (25 patients undergoing GIT surgeries + 25 patients diagnosed with acute pancreatitis);**
- **Control group (CG) – Patients were pooled from neighbouring units (25 patients undergoing GIT surgeries + 25 patients diagnosed with acute pancreatitis).**

Main outcome measures:

Anastomotic dehiscence, wound infection, Respiratory Infection, Day to pass flatus. Day to initiation of bowel sounds, intra-abdominal abscess, length of hospital stay, Serum Albumin, Weight gain, Post op fatigue.

Results & Conclusion

Early enteral feeding was beneficial, associated with fewer complications, and was cost effective in the study.

EARLY ENTERAL FEEDING AND DELAYED ENTERAL FEEDING - A COMPARATIVE STUDY

INTRODUCTION

The human body is a well-oiled machine intended to burn fuel in order to perform work. Nutrients form the fuel for the body, and this comes in three flavours: carbohydrates, proteins, and lipids.

Starvation can adversely affect patients admitted in the surgical wards; more so in the post-operative patients and patients with acute pancreatitis. Those who are kept nil by mouth for extended periods, or have not begun eating by 14 days postoperatively have a significantly higher mortality rate than those who receive nutrition support very early. This, coupled with the fact that malnutrition prevailed among many of the patients admitted in our tertiary health centre (most of them belonging to the lower socioeconomic status), the ramifications of these are overbearing. They eventually lead to a poor outcome. Worldwide studies show that 30% to 50% of hospitalized patients are malnourished, a condition associated with longer hospital stays, higher costs, and increased morbidity and mortality. Patients with malignancies, chronic heart failure or in an immunocompromised state are at particularly high risk. Suppressed immune function can increase risk for nosocomial infections and delayed wound healing. Decreased muscle function can lead to reduced cardiac function and greater difficulty in weaning patients from ventilators. It can also increase susceptibility to respiratory tract infection. Appropriate use of nutritional support can greatly benefit patients in the surgical wards.

Enteral nutrition (EN) means using the GIT to deliver nutrition to the body. In the strictest of the definitions, this means that tubes are used at some level in the gastrointestinal tract for feeding the patient; in this study, oral feeding is also incorporated in the definition, as in the broader sense this route also uses the gastrointestinal tract for nutrition. Parenteral nutrition on the other hand entails the administration of nutrients intravenously. This study will review the administration, rationale and assess the pros and cons associated with the early initiation of enteral feeding.

AIMS & OBJECTIVES

The aim of this study is to evaluate if early commencement of enteral nutrition compared to traditional management (delayed enteral feeding) is associated with fewer complications and improved outcome:

- In patients undergoing elective / **emergency gastrointestinal surgery**;
- In patients with **acute pancreatitis.**

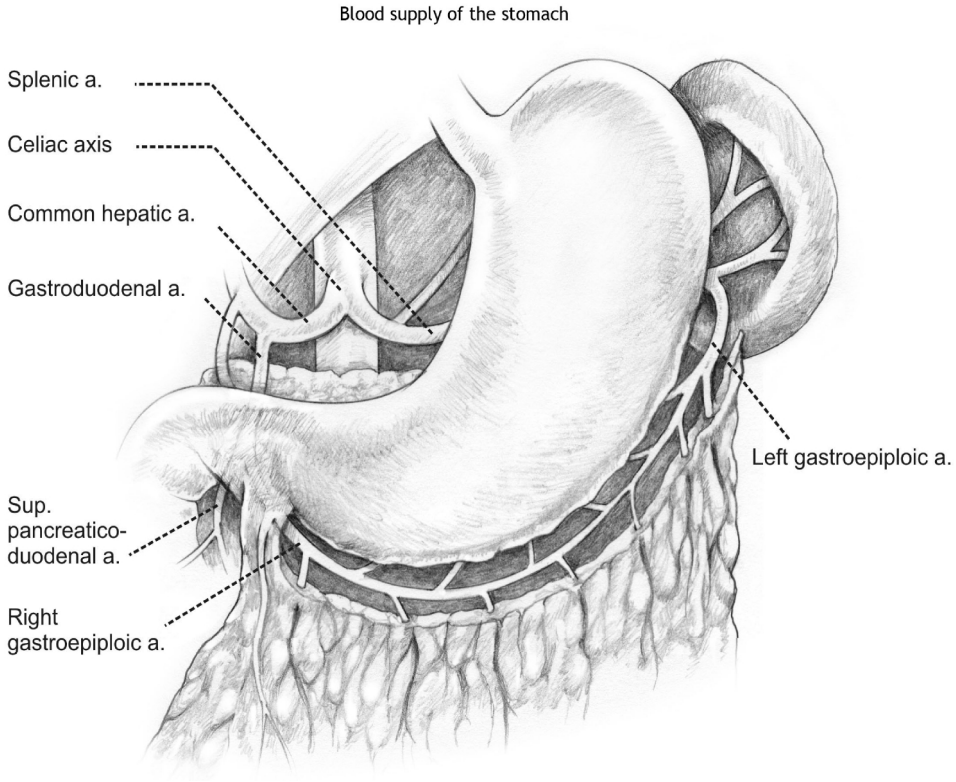
It is also the aim of this study to determine whether a period of starvation (nil by mouth) after gastrointestinal surgery or in the early days of acute pancreatitis is beneficial in terms of specific outcomes.

REVIEW OF LITERATURE

RELEVANT ANATOMY

Vascular anatomy of the Stomach

The stomach is the musculomernbranous expansion of the alimentary canal and is developed from foregut. It is the most dilated part of the digestive tube and lies between the lower end of the oesophagus, and the upper end of the duodenum in the upper part of the abdomen. The arteries of the stomach are the right gastric and right gastroepiploic from the hepatic artery, the left gastroepiploic and vasa brevia from the splenic artery, and the left gastric (coronary) from the celiac axis. These arteries communicate grossly and also by communication between small branches within the gastric wall. Thus the stomach is the most vascularised part of the gut and its arterial supply is so abundant that it is generally considered safe to mobilize the stomach for various anastomosis and also for primary repair in case of gastric perforation.

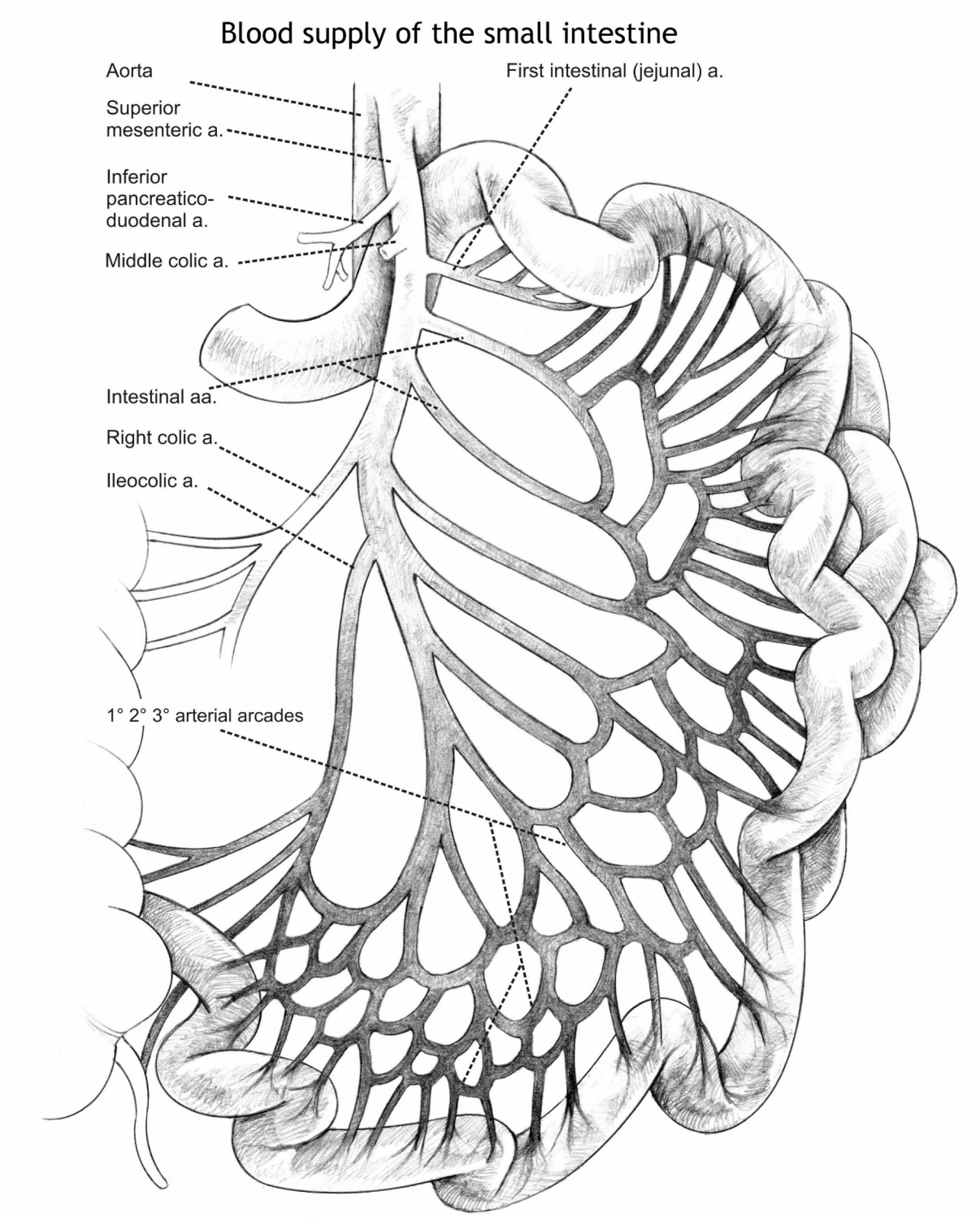


Source: ***Gray's Anatomy, 40th Edition***

Vascular anatomy of the Small Bowel

The small bowel is derived from midgut and originates at the ligament of Treitz and after extending 20 to 30 feet it terminates in the caecum. Proximal part is known as jejunum and the distal part is known as ileum. The wall of the jejunum is thicker than the ileum and its lumen is slightly larger. The blood supply to the small bowel is relatively straightforward and comes directly from the aorta via the superior mesenteric artery. After giving away few branches to pancreas and the middle colic artery, the remaining part of superior mesenteric artery supplies the entire small bowel. The entire blood supply of the small bowel enters at the mesenteric attachment

which is in the retroperitoneum, is 5 to 6 inches in length and runs obliquely from upper left to lower right of abdomen across the aorta and vena cava. The small bowel mesentery is mobile and relatively free to move throughout the peritoneal cavity. Arterial arcades are simpler for jejunum as opposed to more complex arcades for ileum. Meticulous dissection of mesenteric vessels followed by clamping and ligating small arteries and veins with fine and strong suture material or clips is essential to maintain the viability of the cut ends of the small bowel used for anastomosis.



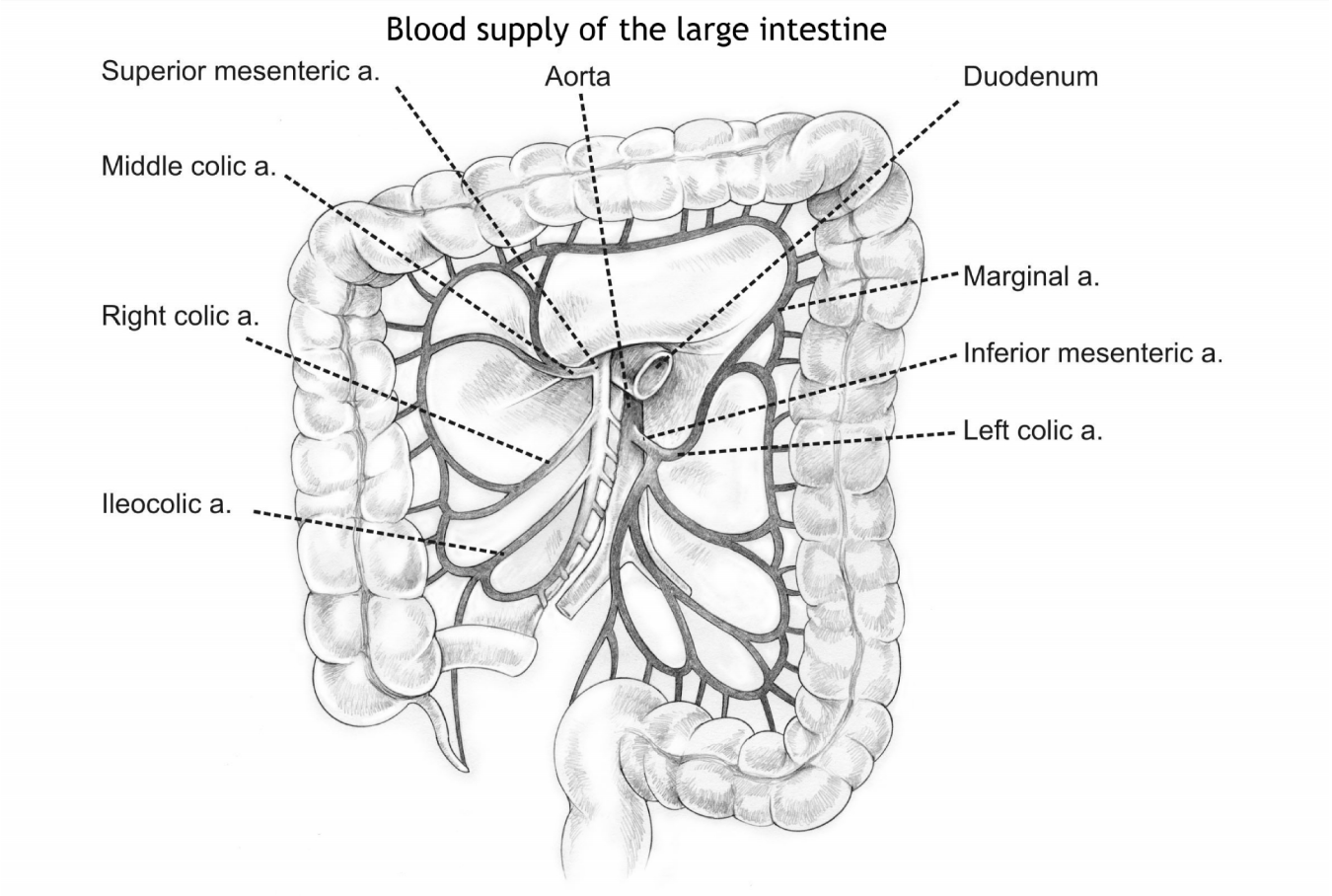
Source: ***Gray's Anatomy, 40th Edition***

Vascular anatomy of the Large Bowel

Large bowel is derived partly from the midgut partly from the hindgut. Midgut portion provides right and transverse colon while hindgut provides the rest of the colon and rectum. The large bowl originates at the ileocaeeal valve in the right lower quadrant of the abdomen where ileum terminates. The total length of the large bowel is 5 to 6 ft and is divided in to right, transverse, left, and sigmoid colon finally dividing into the pelvis to become rectosigmoid and then rectum below the pelvic floor. Right and left colon are devoid of serous coat on their posterior aspect tethering them to the lateral walls of the abdomen with vascular attachments. This fact is of immense importance to the surgeon as wide mobilization of lateral attachments will permit a tension free colonic anastomosis. Colon is identified by the presence of taenia coli on its outer wall. The vascular supply of large bowel comes directly from the aorta via the superior and inferior mesenteric arteries. Right colic and ileocolic branches of the superior mesenteric artery supply the caecum while the middle colic branch supply the transverse colon. The inferior mesenteric artery supplies the rest of the colon and rectum through its left colic, multiple sigmoidal and superior haemorrhoidal branches. The inferior haemorrhoidal artery arising from the internal iliac artery supplies the terminal part of the colon rectum complex. Distal branches of all colic arteries communicate with each other to form the marginal artery which plays a very vital role in vascular simply of the colon. The territory of superior mesenteric artery (SMA) ends at the distal portion of the transverse colon and that of the inferior mesenteric artery (IMA) begins in the region of the splenic flexure. The marginal artery connects these two circulations and forms a continuous arcade along the

mesenteric border of the colon. Vasa recta from this artery branch off at short intervals and directly supply the bowel wall.

The arc of Riolan is a collateral artery first described by Jean Riolan (1580-1657) that directly connects the proximal SMA with the proximal IMA and may serve as a vital conduit when one or the other artery is occluded. It is also known as the meandering mesenteric artery and is highly variabl in size. The flow can either be forwards (IMA stenosis) or retrograde (SMA stenosis) depending on the site of obstruction.



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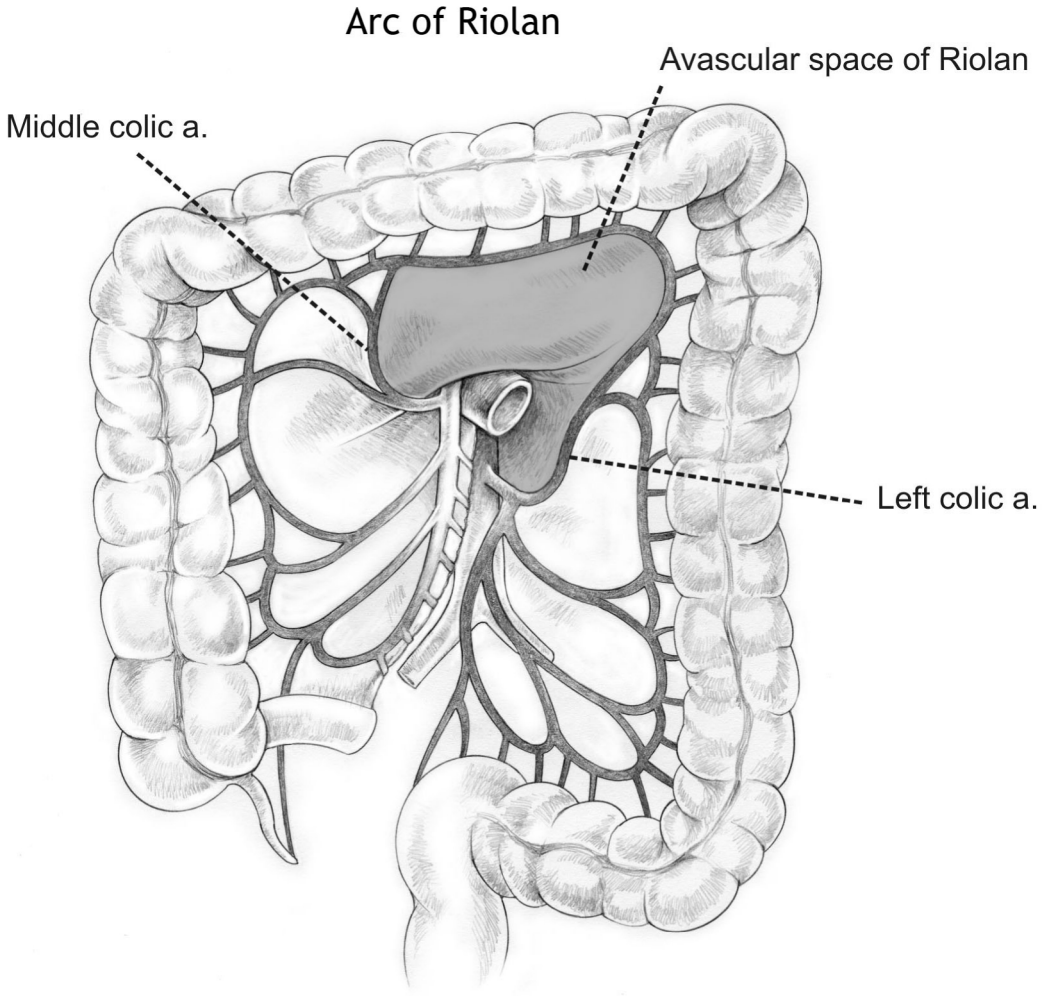

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RELEVANT PHYSIOLOGY

Bowel motility and paralytic ileus are important entities for a surgeon to consider. The motility disorders affecting the GI tract can be better understood by shedding light on the enteric nervous system (EN). The enteric nervous system is incorporated in to the wall of the GIT and is made up of ganglia and primary interganglionic fibre tracts with secondary and tertiary projections to the intestinal musculature. The ENS is arranged in to two plexuses, one overlapping the other anatomically. They are:

1. The myenteric plexus (AKA Auerbach's plexus) located between the longitudinal and the circular muscle layers;
2. The submucosal plexus of Meissner's.

The ENS can be compared to the spinal cord in that it autonomically generates and maintains GI motility under normal physiological conditions. Disturbances in the ENS in certain pathological states can lead to abnormal motility patterns. In addition to this, the submucosal plexus of Meissner's also modulates the local blood flow and GI secretion. The enteric nervous system can be subdivided in to four systems. They are:

1. The Sensory neurons;
2. The Interneurons;
3. The Motor neurons;
4. The Secretomotor neurons.

Prokinetic drugs. such as metoclopramide and erythromycin stimulate the ENS presynaptically and hence increase bowel motility. The central nervous system, via the vagus nerve, controls the autonomic function of the enteric nervous system. This is facilitated by the synapsing of fibres of the vagus nerve with the interneurons. The smooth muscule layer of the GI tract can be considered as a syncytia which can generate action potentials through its intrinsic pacemaker activity. The ENS modulates the excitability of these syncytia.

Postoperative paralytic ileus after an anastomotic procedure or primary repair of perforation is a motility disorder. It occurs as a result of the inhibitory neurons firing continuously and subduing any contractile activity. Such an inhibition paralytic ileus may result due to a multitude of causes including (a) handling of the bowel, (b) intraabdominal abscess, (c) peritonitis, (d) ischaemic bowel, (e) septicemia, (f), metabolic disorders, (g) pneumonia This should serve to remind the surgeon that there is a scientific basis for time-honoured emphasis on meticulous technique and gentle handling of tissues in the conduct of any anastomosis.

Motility of small intestine

Food ingested and digested up to the stomach reaches the duodenum and is propelled further through the small bowel by a series of muscular contraction called “peristalsis”. They are intestinal contractions passing from proximal to distal at a rate of 1 to 2 cm/sec. This facilitates the movement of intestinal chyme through the rest of the bowel. The rhythmicity of these peristaltic waves varies in the fed and fasting states. Duodenum is thought to be the site of origin of the so called “pacemaker

potentials", which initiate a series of contractions in the fed state. This is supposed to propel the food through the small bowel. In the fasting state, the rythmicity changes. The bowel is periodically swept by cyclical contractions that move distally along the intestine every 75 to 90 minutes. These contractions are initiated by the migrating myoelectric complex (MMC). Both humoral and neuronal pathways control the MMCs'.

The small bowel is also innervated by the extrinsic system; mainly by the vagus (parasympathetic) and the sympathetic nervous system. Vagal stimulation has two main effects on the small bowel: one is cholinergic, which is excitatory and the other peptidergic, which is inhibitory. Sympathetic activity inhibits motor function whereas parasympathetic activity stimulates it. The most important intestinal hormone that affects the motility of the small bowel is 'motilin'. The plasma level of motilin is at the maximum when the MMCs are present.

Motility of large intestine

Fermentation in the colon is made possible by its distinctive morphology. The colon can be divided into three anatomic segments; the right colon, the left colon, and the rectum. The right colon is the fermentation chamber of the human GIT, with the caecum being the colonic segment where bacteria are most metabolically active. The left colon is a site of storage and dehydration of stool. Transit through the colon is controlled by the autonomic nervous system. Parasympathetic nervous fibres supply the colon via the vagi and the pelvic nerves. Nerve fibres reaching the colon arrange themselves in several plexuses, similar to the ones in the small intestine; the

subserosal Auerbach's, the submucosal (Meissner's) and the mucosal plexuses. The neurons of the myenteric plexus concentrate along the taeniae but sparsely between them where the longitudinal muscle layer is thin. Sympathetic nerve fibers originate in the superior and inferior mesenteric ganglia and reach the colon by way of perivascular plexuses.

The motility pattern is different in the three anatomic segments. In the right colon, antiperistaltic or retropulsive waves generate retrograde flow of colonic contents back to the caecum. In the left colon, contents are propelled caudad by tonic contactions separating them into a series of globular masses. A third type of contraction called mass peristalsis is interspersed with the propulsive and retropulsive contractions and occurs at varying intervals, more frequently after meals. Each mass peristaltic contraction is able to advance a column of colonic contents through one third of the colonic length.

After the intake of a meal, there is an increase in the bursts of action potentials in the colon, which peaks at around fifteen minutes after the meal; the effect of which is an increase in the colonic tone. On comparing, this "postprandial contractility" is more in the sigmoid colon than in the transverse colon. This colonic motility that ensues after a meal is called the 'gastrocolic reflex'.

RATIONALE FOR USING EARLY ENTERAL NUTRITION

Mucosal Atrophy

Historically, it was thought that the GIT was quiescent following surgical intervention or after an episode of pancreatitis, and that the primary role of the gut was digestion, absorption, and secretion. It is now evident that the gut is an important metabolically active organ and plays a vital role in nutrient transport, exposure of nutrients to absorptive mucosa, prevention of stasis and bacterial overgrowth, as well as immune regulation.

The idea here was simple. Non-usage of the GIT for a prolonged time as a result of starvation could lead on to gut mucosal atrophy; just like any other disuse atrophy. Food in the intestinal lumen was thought to be critical to normal function and cell growth of gut mucosa.

Bowel rest and TPN in rats have been found to cause gut atrophy within days, an outcome thought to be the result of lack of functional stimulation as well as reduced pancreatic and biliary secretions. Pironi et al. reported significant changes in morphologic and cytoproliferative patterns of duodenal mucosa with the administration of long-term TPN. Similar data from Groos et al. show decreased epithelial cell turnover, and extracellular matrix on prolonged starvation in humans. The clinical repercussions of these changes are obvious. As a result, efforts have been focused on using the GI tract whenever possible.

Bacterial Translocation

The jury is out on this; if starvation causes microbial translocation; and even if translocated, of what significance is that in causing post-operative sepsis.

Animal studies have suggested an association between bacteria translocation and postoperative sepsis. O'Boyle et al. have reported a relation between microbial translocation from the gut and postoperative sepsis, but not mortality. Data from human studies have shown a relation between gut microflora and nosocomial infections, once the gut is put to rest for extended period. Other data reports have shown no spatial or temporal relation between the translocation of gut bacteria and the presence of post-op sepsis. Sedman et al. have suggested that prevalence of bacterial translocation is the same in patients receiving either TPN or EN.

Nevertheless it must be known that the prevalence of microbial translocation is nearly 15% in patients undergoing elective surgeries and higher in patients undergoing emergency surgeries or are in an immunocompromised state.

Infectious Complications

A recent meta-analysis examined the relation between nutritional interventions, complications, and death rates. 27 studies with 1,828 patients have shown a 34% decreased risk of infection with EN compared with TPN. EN was associated with a reduced risk of infection immaterial of the nutrition status, presence of malignancy, or the quality and year of study. These findings were also independent of catheter sepsis analysis. The increased risk of infection may be related in part to a higher incidence of

hyperglycemia in this population. Excess glucose load and stress response in TPN-fed patients may lead to impaired immune responses that contribute to greater risk of infection.

Noninfectious Complications

A comparison of noninfectious complications showed a 36% greater risk for EN compared with TPN. Such complications included TPN-related and EN-related technical (caused by tube or catheter insertion) and mechanical problems (dislodged or occluded tube or catheter); aspiration; diarrhea; vomiting; fistula at the catheter or tube site; and hyperglycemia. Many of the complications associated with EN (e.g., diarrhea or abdominal distention) occur frequently, but are considered less severe than catheter sepsis. Because EN and TPN are not without risks, their advantages and disadvantages must be carefully weighed before the initiation of either type of nutrition support.

Cost

Data show that EN is less expensive to administer than TPN. Costs include access devices, insertion, solutions, delivery hardware, laboratory monitoring, clinical monitoring, and complications.

AN IDEA ABOUT "STARVATION"

How long can patients be allowed to do without some form of nutrition is a matter of debate for quite some time now. A study was done on the voluntary starvation deaths of Irish Republican Army (IRA) members in Northern Ireland; patients who drank water but no nutritional liquids died after about 2 months to 10 weeks. The common cause of death was pneumonia, as the individuals could no longer clear secretions because of muscular weakness. The estimated loss of lean body mass was 30%. In the absence of infection, nitrogen breakdown decreases dramatically after 4 or 5 days, and fat and ketosis takes over so that muscle protein breakdown is minimized. Nonetheless, the breakdown of muscle is not zero, and ultimately the person dies. The rule of thumb is that patients who are well nourished probably can manage about 10 to 14 days of complete starvation, or starvation with a little bit of glucose, before serious deterioration in muscle mass, ability to clear secretions, and mood take place. In octogenarian patients this period is about 5 days, and in septuagenarians it is about 7 or 8 days. One should not allow a period of starvation for a patient of any age; there should be intervention as soon as it is clear that these patients will not be eating for a period of time.

As for the type of nutritional support, it is now generally accepted that one should use the gut if one can. Even if one cannot get the total amount of calories in by gut, as little as 20% will allow the patient to derive the benefit of enteral nutrition. What this benefit might be is a matter of some controversy. Gut as the engine of multiple organ failure has been disproved; but the hypothesis is still mentioned in

literature. There are probably more than 28 studies of gut sterilization in intensive care patients showing that there is no difference in outcome except in pneumonitis (probably because of aspiration) as far as multiple organ failure. The benefits of enteral nutrition probably are reflected in hepatic protein synthesis in which the protein equivalences are absorbed by the portal vein and go to the liver for acute-phase protein synthesis. This is especially evident in burns patients, for which excellent data have been obtained by Alexander et al.

STRESS METABOLISM

Alterations in metabolism due to physiologic stress share similar patterns with simple starvation. Regardless of the stimulus, our conserved response to stress is the same—catabolic shifts mobilize energy stores in order to prepare us to "fight or flight.". The two stresses discussed here are,

1. Simple starvation;
2. Physiological stress.

Simple Starvation

The primary sources of fuel in an adult during a short period of starvation (< 5 days) are body fat and muscle protein, with fat constituting the most abundant source of energy. The following table shows the breakdown of body fuel reserves in a 70 kg man.

Body Fuel Reserves in a 70 kg Man			
A. COMPONENT	**MASS (kg)**	**ENERGY (kcal)**	**DAYS AVAILABLE**
Water and minerals	49	0	0
Protein	6.0	24,000	13.0
Glycogen	0.3	1200	0.4
Fat	15.0	140,0000	78.0
Total	**70.3**	**165,200**	**91.4**

Source: EBERLEIN, T. J. (2012). The Washington manual of surgery

One can derive from the table that the body contains about 0.3 kg of glycogen. Of this, around 100 g are stored in the liver. The rest of the glycogen are stored within skeletal muscle, cardiac muscle, and smooth muscle cells. But this greater proportion of glycogen in muscle cells is not available for systemic use because of the simple fact that muscle cells are deficient in glucose-6-phosphatase; nevertheless this glycogen is available for the energy needs of muscle cells. Hence after an overnight fast, liver glycogen is rapidly depleted during glycogenolysis, as glucagon responds to falling serum glucose levels. Eventually all the carbohydrate stores are exhausted after 24 hours.

Like previously stated, for the first few days during starvation, caloric needs are met by fat and protein degradation. Most of the protein is from breakdown of

skeletal and visceral muscle, which is converted to glucose via hepatic gluconeogenesis. The precursors of gluconeogenesis is as follows:

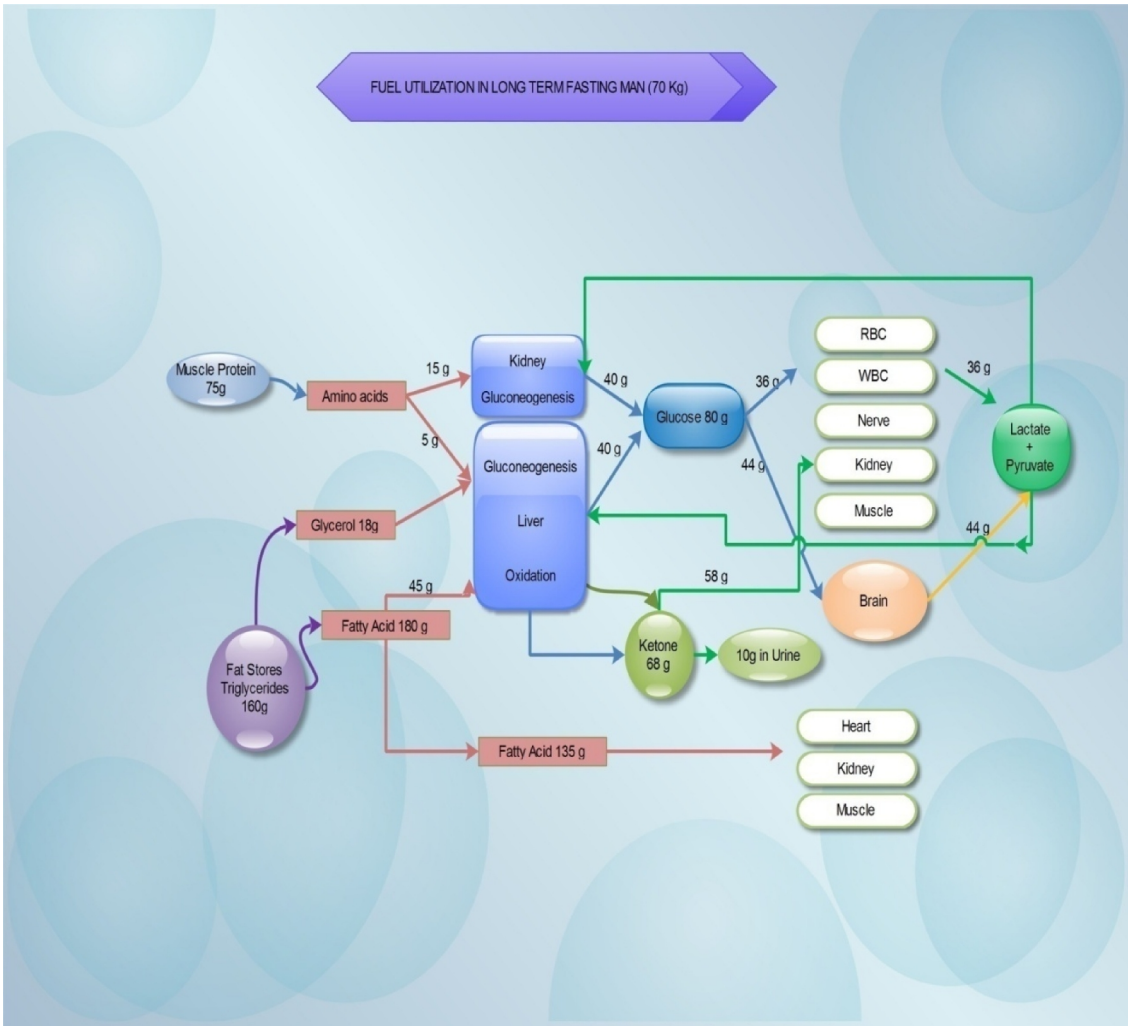
1. Lactate from glycolysis in skeletal muscles, red cells and leukocytes;
2. Glycerol (part of the triacylglycerol molecule) from oxidation of odd chain fatty acids;
3. Amino acids from protein breakdown. (Significant proportion of protein must be broken down daily (around 75 g/d for a 70-kg adult) to provide the necessary amino acid substrate for hepatic gluconeogenesis).

The brain preferentially uses this endogenously produced glucose, with the remainder consumed by red blood cells and leukocytes. The kidneys also participate in gluconeogenesis and with prolonged starvation can account for up to half of the glucose production.

Within approximately 10 days of starvation, the brain adapts and uses fat in the form of ketoacids as its fuel source. Produced by the liver from free fatty acids, the use of ketoacids has a protein-sparing effect. In an adult, around 160 g of free fatty acids and glycerol can be mobilized from adipose tissue per day; hence fat stores can last for about 11-12 weeks.

Energy Equivalent of Substrate Oxidation					
SUBSTRATE	**O2 CONSUMATED (L/g)**	**CO2 PRODUCED (L/g)**	**RESPIRATORY QUOTIENT**	**KCAL/G**	**RECOMMENDED DAILY REQUIREMNT**
Glucose	0.75	0.75	1.0	4.0	7.2 g/kg per day
Dextrose	-	-	-	3.4	-
Lipid	2.0	1.4	0.7	9.0	1.0 g/kg per day
Protein	1.0	0.8	0.8	4.0	0.8 g/kg per day

Source: EBERLEIN, T. J. (2012). The Washington manual of surgery



Physiologic stress

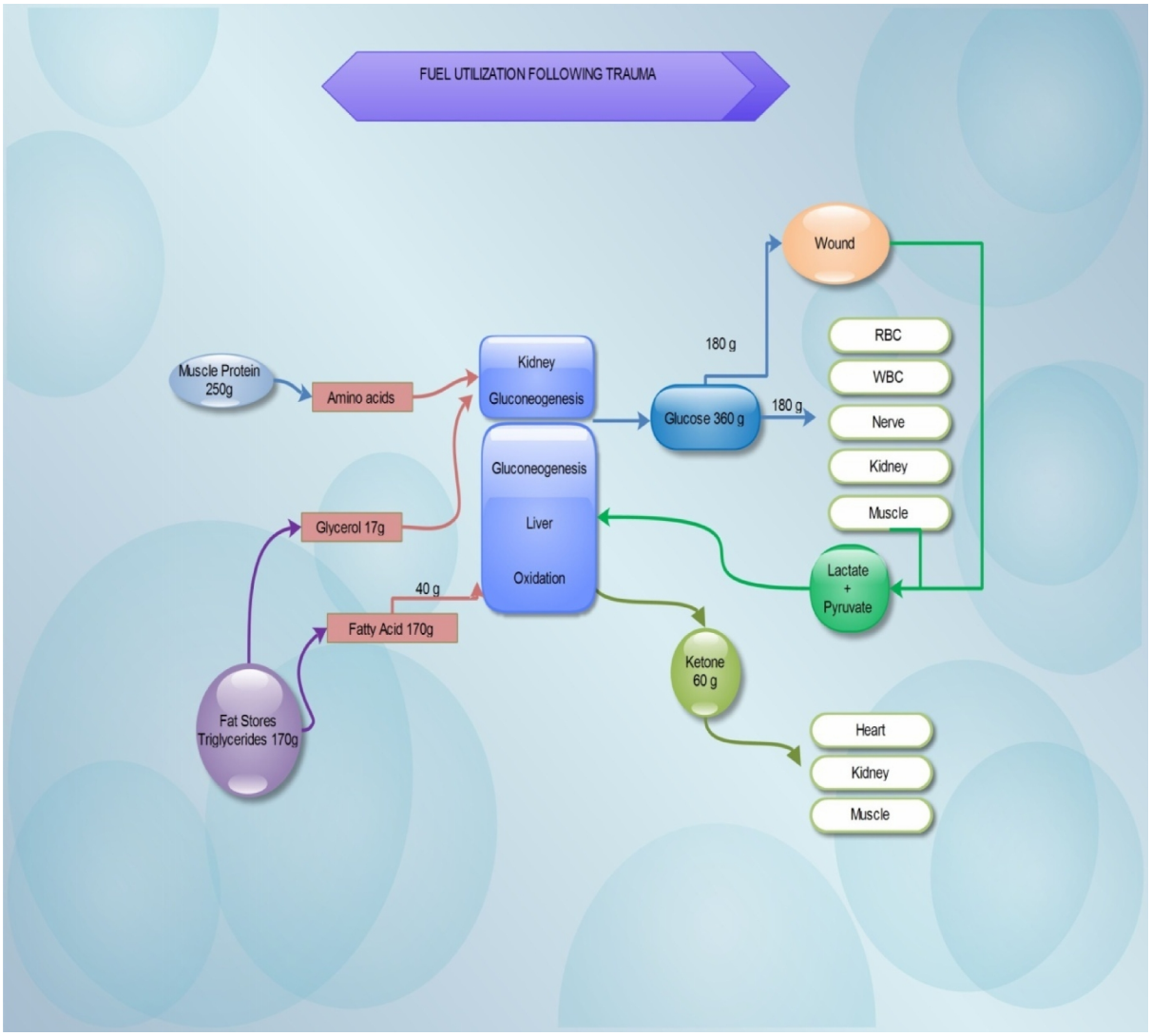
A healthy adult requires approximately 22 to 25 kcal/kg per day of macronutrients. In stressful states, this demand can go up as high as 40 kcal/kg/day. An example would be the metabolism in burns patients.

The interaction of metabolic and endocrine responses that result from major operation, trauma, or sepsis can be divided into three phases:

- **Catabolic phase**. After major injury, the metabolic demand is dramatically increased, as reflected in a significant rise in the urinary excretion of nitrogen (beyond that seen in simple starvation). Following a major surgical procedure, protein depletion inevitably occurs because patients are commonly prevented from eating in addition to having an elevated basal metabolic rate. The hormonal response of physiologic stress includes elevation in the serum levels of glucagon, glucocorticoids, and catecholamines and reduction in insulin;
- The **early anabolic phase** is also called the corticoid withdrawal phase as the body shifts from catabolism to anabolism. The timing of this event is variable, depending on the severity of stress, and ranges from several days to several weeks. The period of anabolism can last from a few weeks to a few months, depending on many factors, including the ability of the patient to obtain and use nutrients and the extent to which protein stores have been depleted. This phase is marked by a positive nitrogen balance, and there is a rapid and progressive gain in weight and

muscular strength. The total amount of nitrogen gained is equivalent to the amount lost in the catabolic phase; however, the rate of repletion is much slower than the rapid rate of protein depletion after the original insult;

- The **late anabolic phase** is the final period of recovery and may last from several weeks to months. Adipose stores are replenished gradually and nitrogen balance equilibrates. Weight gain is much slower during this period than in the early anabolic phase due to the higher caloric content of fat—the primary energy stores deposited during the early anabolic phase—as compared to protein.



RELEVANT ANATOMY OF THE PANCREAS

Pancreas (pan: All & kreas: flesh)

The pancreas is an entirely retroperitoneal structure. It is divided into four regions: the head/uncinate, neck, body, and tail. The head of the pancreas abuts the C loop of the duodenum and extends obliquely to the neck, anterior to the mesenteric vessels and portal vein. The neck then extends laterally into the body, which is

generally accepted to begin at the left border of the superior mesenteric vein (SMV), lying posterior to the stomach and anterior to the splenic vessels. The pancreas then culminates in the tail that is associated with the splenic hilum anterior to the left adrenal gland. The pancreas receives its blood supply from both the celiac trunk and the superior mesenteric artery (SMA). The arterial supply of the pancreatic head is provided by the inferior pancreaticoduodenal arteries (from the SMA) and the superior pancreaticoduodenal arteries (from the gastroduodenal artery). The tail receives its arterial supply from branches of the splenic artery. Venous drainage is primarily by the pancreaticoduodenal veins, which drain into the portal vein. The pancreas has two ducts: the main pancreatic duct, called the Duct of Wirsung, which arises in the tail and traverses the length of the pancreas to terminate at the papilla of Vater within the wall of the duodenum; and the Duct of Santorini, which is much smaller and arises from the lower part of the head, terminating separately at the lesser papilla.

Acute Pancreatitis

Acute pancreatitis is an inflammatory process of variable severity. Approximately 80% of cases of acute pancreatitis are self-limited and associated with mild transitory symptoms that do not cause fulminant morbidity or mortality. By contrast, 20% of patients develop a severe form of acute pancreatitis that is associated with a mortality rate as high as 40% (Nat Clin Pract Gastroenterol Hepatol. 2005;2:473). The exact mechanism by which various etiologic factors induce acute pancreatitis is unclear. However, most agree that the initial insult is unregulated

activation of trypsin within pancreatic acinar cells, leading to autodigestion and an inflammatory cascade that may progress to SIRS (Lancet. 2008;371(9607):143–152).

Prognosis

Because the associated mortality of fulminant acute pancreatitis approaches 40% and randomized studies have shown that early aggressive supportive care improves outcomes, attempts have been made to identify clinical parameters that predict patients at higher risk of developing severe outcomes.

Ranson criteria constitute the most frequently utilized predictor of mortality associated with acute pancreatitis. The limitation of this assessment tool is that a score cannot be calculated until 48 hours after admission.

CTSI is a prognostic scale based on CT findings, including peripancreatic fluid collections, fat inflammation, and extent of pancreatic necrosis was originally described by Balthazar et al. (Radiology. 1994;174:331–336) and then modified to a simpler model (Am J Roent. 2004;183:1261–1265). The usefulness of this criterion is limited to patients with normal renal function able to undergo contrast-enhanced CT, and by the fact that many patients with limited disease do not undergo CT imaging, potentially skewing study results (see Tables 13-2 and 13-3).

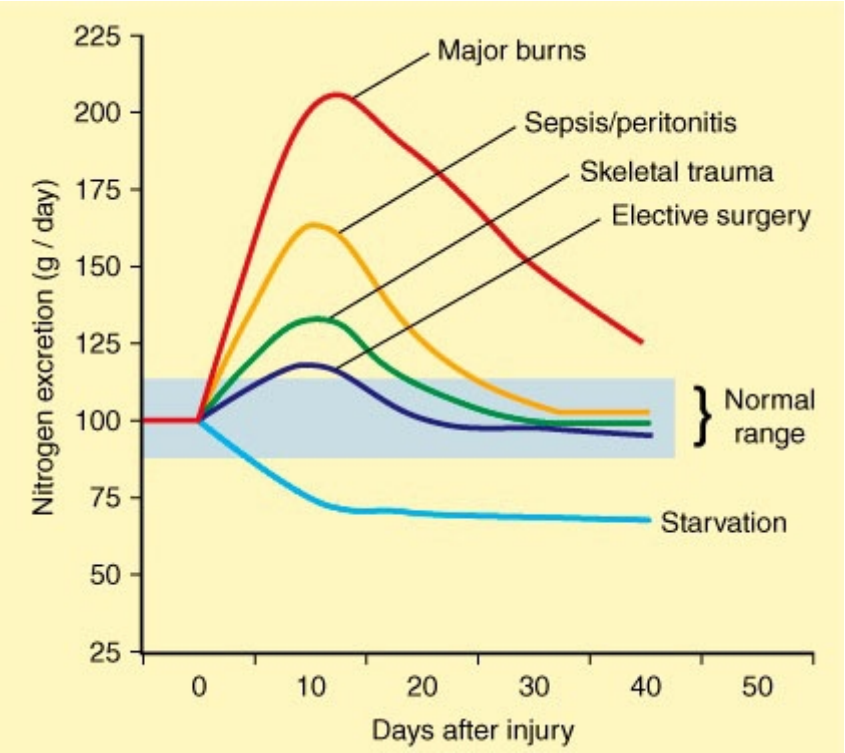
The Glasgow scoring, like Ranson criteria, requires 48 hours to prognosticate patients and is based on clinical and laboratory values.

The Acute Physiology and Chronic Health Evaluation (APACHE) II score was developed in 1985 for assessing critically ill patients and incorporates physiology, age, and chronic health. The major benefit of the APACHE II score is that it can be calculated at admission and updated daily to allow continual reassessment. However, the APACHE II score is somewhat cumbersome and difficult to calculate that limits its everyday use.

Multiple Organ Dysfunction Score (MODS) and Sequential Organ Failure Assessment (SOFA) have been shown to be important predictors of disease severity in critically ill patients and have been extended to patients with severe acute pancreatitis and are predictive of mortality and development of complications (Br J Surg. 2009;96(2):137–150).

Comments

The incidence of pancreatitis is increasing worldwide, and this has greater implications in a country like India, where the consumption of alcohol is increasing at a steady state. Severe cases of pancreatitis are associated with development of sepsis and multi organ dysfunction. Patients have traditionally been kept NPO to minimize pancreatic stimulation and decrease its subsequent inflammation. This practice is known to lead on to intestinal ischaemia, bacterial translocation, and potential sepsis. A metaanalysis of 291 patients has revealed that patients with acute pancreatitis who received some form of enteral nutrition had significantly reduced infectious complications although no differences were observed in mortality.



Source: Schwartz's principles of surgery

ESTIMATION OF ENERGY NEEDS

The basal energy expenditure is calculated by using the 'Harris-Benedict equation' (in kilocalories per day):

The formula for men is as follows:

66.4 + [13.7 × weight (kg)] + [5 × height (cm)] - [6.8 × age (years)].

The formula for women is as follows:

65.5 + [9.6 × weight (kg)] + [1.7 × height (cm)] - [4.7 × age (years)].

These equations give a reasonable estimate of the energy requirements in up to 80% of hospitalized patients. The actual caloric need vary depending upon the the type of stress, and is calculated by using a multiplier factor as shown in the table. Most stressed patients require 25 to 35 kcal/kg/day.

Disease Stress Factors Used in Calculation of Total Energy Expediture	
CLINICAL CONDITION	**STRESS FACTOR**
Starvation	0.80 – 1.00
Elective operation	1.00 – 1.10
Peritonitis or other infections	1.05 – 1.25
Pancreatitis	1.30 – 1.80
Adult respiratory distress, syndrome or sepsis	1.30 – 1.35
Cardiopulmonary disease (uncomplicated)	0.80 – 1.00
Cardiopulmonary disease with dialysis or sepsis	1.20 – 1.30
Cardiopulmonary disease with major surgery	1.30 – 1.55
Acute renal failure	1.30
Liver failure	1.30 – 1.55
Liver transplant	1.20 – 1.50

Source: Schwartz's principles of surgery

ESTIMATION OF PROTEIN NEEDS

The appropriate calorie-nitrogen ratio is approximately 150:1 (calorie:protein ratio of 24:1). In the absence of severe renal or hepatic dysfunction, approximately 1.5 g protein per kilogram body weight should be provided daily.

Estimated Protein Requirement in Various Disease States	
CLINICAL CONDITION	**PROTEIN REQUIREMENTS (G/KG IDEAL BODY WEIGHT PER DAY)**
Healthy, nonstressed	0.80
Bone marrow transplant	1.40 – 1.50
Liver disease without encephalopathy	1.00 – 1.50
Liver disease with encephalopathy	0.50 – 0.75 (advance as tolerated)
Renal failure with/without dialysis	0.60 – 1.30
Pregnancy	1.30 – 1.50
Simplified Estimates	1.30 – 1.50
Mild metabolic stress (elective hospitalization)	1.00 – 1.10
Moderate metabolic stress (complicated postoperative care, infection)	1.20 – 1.40
Severe metabolic stress (major trauma, pancreatitis, sepsis)	1.50 – 2.50

Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

ENTERAL FORMULAS

Quite a few enteral formulas are available for both oral and tube feeding. They differ from one another based on the composition and proportions of the macro and micronutrients. These formulas have now evolved from simple foods prepared in a blender and then fed to the patients to much more disease specific formulas. Nevertheless these 'blender preparations' are still useful in a health system like ours, where cost effectiveness is an issue.

There are three major groups of enteral formulas:

1. Polymeric-balanced
2. Monomeric (elemental), and
3. Disease-specific.

Polymeric-Balanced Formulas

Polymeric formulas are also known as standard formula-feeds. They are well tolerated and can be used in patients with a normal GI tract. When administered as

prescribed, they provide nutritionally complete and balanced diets that include most required micronutrients and macronutrients. The breakdown of the macronutrients is as follows

1. 50% to 55% carbohydrate(range from simple sugars to complex carbohydrates)
2. 15% to 20 % protein (Whole protein), and
3. 30% fat (Vegetable oils high in long-chain triglycerides (LCTs))

They generally yield 1 kcal/mL. Calorie-dense formulas (1.5 to 2 kcal/mL) are also available, but should be reserved for patients who require high energy intake or fluid restriction. Normal levels of pancreatic enzymes are required to digest the proteins in them. Hence they have a restricted use in pancreatitis.

Polymeric-balanced formulas are the least expensive among the commercially available options for EN. In up to 90% of surgical patients, they are the formula of choice.

Monomeric (Elemental) Formulas

Elemental formulas are more expensive than polymeric alternatives. They use free amino acids or small chain peptides as protein sources and are easy to digest. Elemental formulas are usually low in fat or high in MCTs. They are typically prescribed for patients with maldigestion and malabsorption; e.g., pancreatitis, critical illness, short-gut syndrome, enterocutaneous fistulas, and diarrhea. In patients

undergoing routine gastrointestinal operations, elemental formulas offer no benefit over polymeric formulas

Modular Supplements

In cases in which commercially available enteral formulas may not be optimal, patients may benefit from modular feeding systems. Modular supplements contain single or multiple nutrients (protein, carbohydrate, and/or fat) that can be added to liquid enteral formulas.

Disease-Specific Formulas

Disease-specific formulas modify nutrient profiles to achieve desired outcomes, such as immune enhancement, or address specific disease states, such as renal, hepatic, and pulmonary states.

Immune-Enhancing Formulas

Various enteral formulas contain substrates with immune-modulating properties, e.g., glutamine, arginine, omega-3 fatty acids, nucleotides, selenium, and vitamins A, C, and E. Studies suggest that these formulas reduce the incidence of infectious complications, decrease ventilator time, shorten hospital and intensive care stays, and reduce patient hospital costs. At the same time, the use of immune-enhancing formulas in severely ill patients has been found to increase mortality, lengthen stays in hospital and intensive care units, extend ventilator time, and raise treatment costs. Data indicate that this type of diet benefit patients who are undergoing elective GI surgeries and are moderately to severely malnourished. It is also useful in

patients with severe blunt and penetrating trauma to the abdomen, and some critically ill patients.

Renal Diets

Patients with stable chronic renal failure typically need energy-dense, low-protein diets, and those undergoing hemodialysis require high-protein diets. Use of essential amino acids or branched chain amino acids (BCAAs) shows no clear benefit in efforts to meet caloric needs and avoid protein load. Studies have yet to show clinical efficacy of renal failure formulas; therefore, their use should be limited to efforts to avoid dialysis in cases of acute renal failure, or to reduce dialysis requirements. With renal dysfunction, metabolism of certain vitamins and minerals (e.g., folic acid, pyridoxine, calcium, and vitamins A, C, and D) and excretion of electrolytes (e.g., potassium, magnesium, and phosphorus) may be impaired. It may be necessary to adjust the intake.

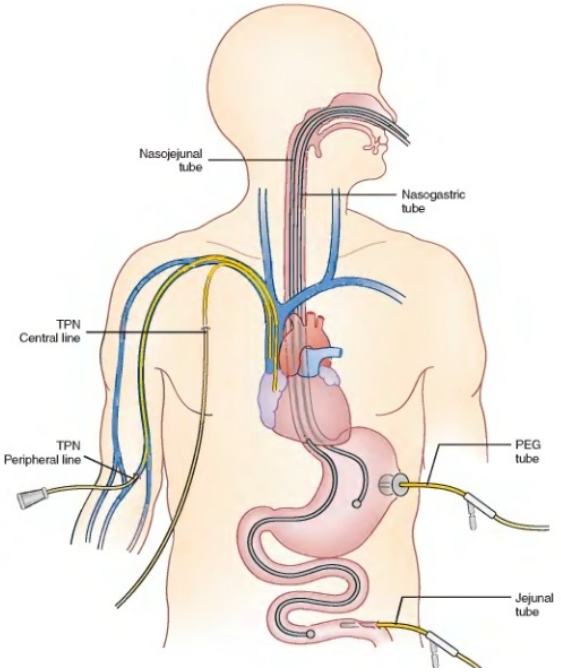
Hepatic Formulas

Hepatic enteral formulas contain large amounts of the branched chain amino acids (BCAA), namely leucine, isoleucine and valine. They also incorporate aromatic amino acids (AAAs) in them, namely phenylalanine, tyrosine, and tryptophan. The formulas are designed to reduce AAAs in the blood–brain barrier by normalizing AAA to BCAA ratios in the plasma of patients with hepatic encephalopathy. Data suggest that BCAA supplements provide necessary nitrogen intake to some protein-intolerant patients with no adverse effect on mental state, and perhaps even improvement. Because the use of BCAA supplements remains controversial,

administration of formulas for hepatic failure should be limited to patients with hepatic encephalopathy who do not respond to standard treatments.

Pulmonary Diets

Patients with pulmonary insufficiency exhibit carbon dioxide (CO2) retention and oxygen (O2) depletion. Pulmonary formulas are low in carbohydrates and high in fats (approximately 50% of calories); they attempt to decrease respiratory quotient and minimize CO2 production and retention. It is prudent to avoid carbohydrate overfeeding in patients with acute and chronic lung disease until data from clinical trials of adequate sample size become available. Critically ill patients who have difficulty weaning from ventilators may require a short-term decrease or discontinuation of feeding.



ENTERAL ACCESS AND INSERTION/PLACEMENT

A knowledge about the patient's GI tract anatomy and function, the duration of feeding that one expects in the patient, and the potential risk for aspiration, will give an idea about selecting the appropriate route of access.

Gastric feeding is the preferred approach. Gastric access is physiologically accessible, convenient, and makes feeding easy to begin. However, it requires the patient be conscious, has intact gag and cough reflexes and adequate gastric emptying.

Types of Access

The various routes of accesss to the GIT can be broadly divided in to the following:

1. Nasoenteric, includes nasogastric, nasoduodenal and nasojejunal;
2. Gastrostomy techniques;
3. Jejunostomy techniques.

Nasoenteric

Nasoenteric, the most commonly used enteral access, is indicated for short-term use (<4 weeks). As mentioned before, they can be inserted into different levels, namely the stomach, duodenum, or jejunum.

Complications

Complications associated with nasally inserted tubes include ulcers on the walls of the nasopharynx, loco-regional infection causing sinusitis and otitis, and injury to the vocal cords leading hoarseness of voice or even stridor. Prolonged compression of the nasal septum by a large stiff feeding tube might cause septum necrosis. Feeding tubes of a smaller calibre, made from silicone or polyurethane which are soft, smooth, and more flexible, can be used instead of larger, stiffer tubes. Smaller calibre tubes are well tolerated for 3 to 4 weeks and they decrease the risk of nasal tissue necrosis.

Small Bowel

This route of access is preferable in patients who are at risk of recurrent aspiration of gastric contents (e.g., severe GORD, esophageal dysmotility, gastroparesis, and gastric outlet obstruction). It is also indicated when early postoperative feeding is planned after major abdominal surgery.

Advantages

In addition to lessening the aspiration risk, postpyloric feeding also minimizes the stimulation of pancreatic enzyme secretion. Therefore, it can be helpful in critically ill patients with acute pancreatitis and those at risk for gastric motility dysfunction.

Disadvantages

Major disadvantages of postpyloric feeding include difficult tube placement, maintenance of proper positioning, and clogging.

Placement Methods

Several methods can be used for postpyloric tube placement. The bedside method entails placing the tube in the stomach and allowing it to gradually migrate into the small intestine (with aid from the peristaltic waves). Spontaneous migration of a simple nasojejunal tube to the small bowel occurs in 33% of insertions. Recent studies show high success rates when pH monitoring is used with jejunal tubes. Nasoenteric tubes with weighted tips do not facilitate small bowel placement. However, data suggest that use of prokinetic agents (e.g., erythromycin or metoclopramide) prior to tube placement can help position small nasoenteric tubes.

When blinded methods fail, endoscopy-, fluoroscopy-, and sonography-guided transpyloric placement are frequently used. These techniques have high success rates for initial placement. O'Keefe et al. reported a 90% success rate with transnasal endoscopic placement of double-lumen gastric aspiration, jejunal feeding tubes in the intensive care units. In that study, a video endoscope was used to place a guidewire through the nose, terminating beyond the ligament of Treitz. After withdrawal of the endoscope, a double-lumen gastric aspiration, jejunal feeding tube was passed over the wire. However, tubes often migrate back to the stomach or dislodge, necessitating repeated insertions. The position of tubes should be confirmed by chest radiograph before use for feeding or drug administration.

Gastrostomy

Long-term (>4 weeks) EN calls for permanent access to the gastrointestinal tract. Insertion of tube enterostomies can be performed with endoscopic or fluoroscopic assistance, or surgically. The two most common sites for placement of long term access include the stomach and jejunum.

Contraindications to Gastrostomy

Contraindications to gastrostomy include gastroesophageal reflux disease, previous upper abdominal surgery, gastric outlet obstruction, large gastric varices, malignant or infiltrative disease of the stomach, gastric dysmotility, and massive ascites. Coagulopathy must be corrected before percutaneous placement of a feeding tube.

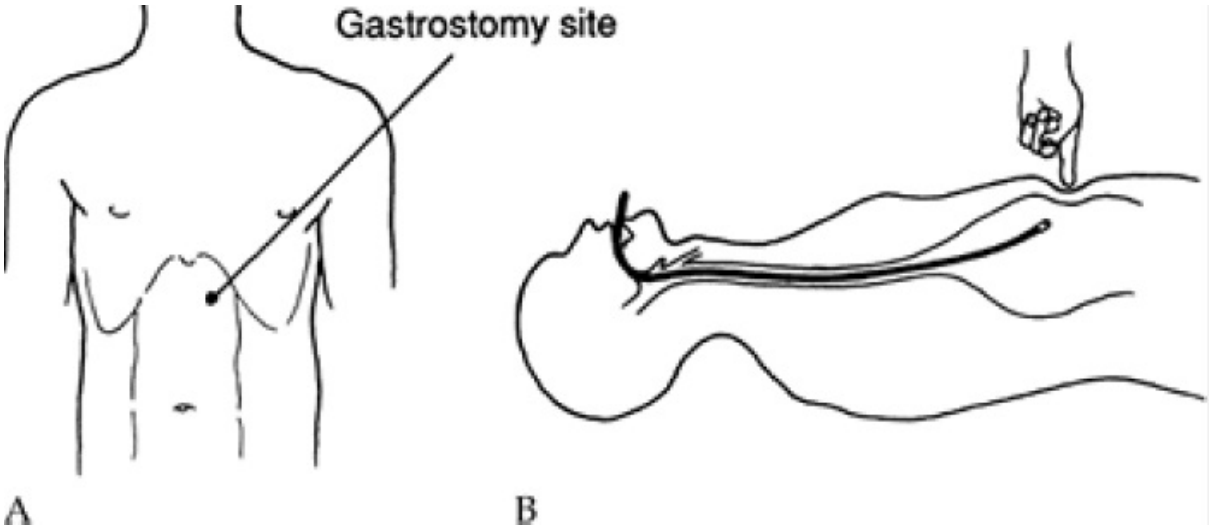
Percutaneous Gastrostomy

Advantages of percutaneous placement of gastrostomy tubes include insertion without general anesthesia, feeding soon after placement, and decreased morbidity and mortality. Most hospitals prefer percutaneous endoscopic gastrostomy (PEG) rather than surgical placement because it is relatively fast, simple, safe, and the cost is low.

There are several ways to place PEG tubes:

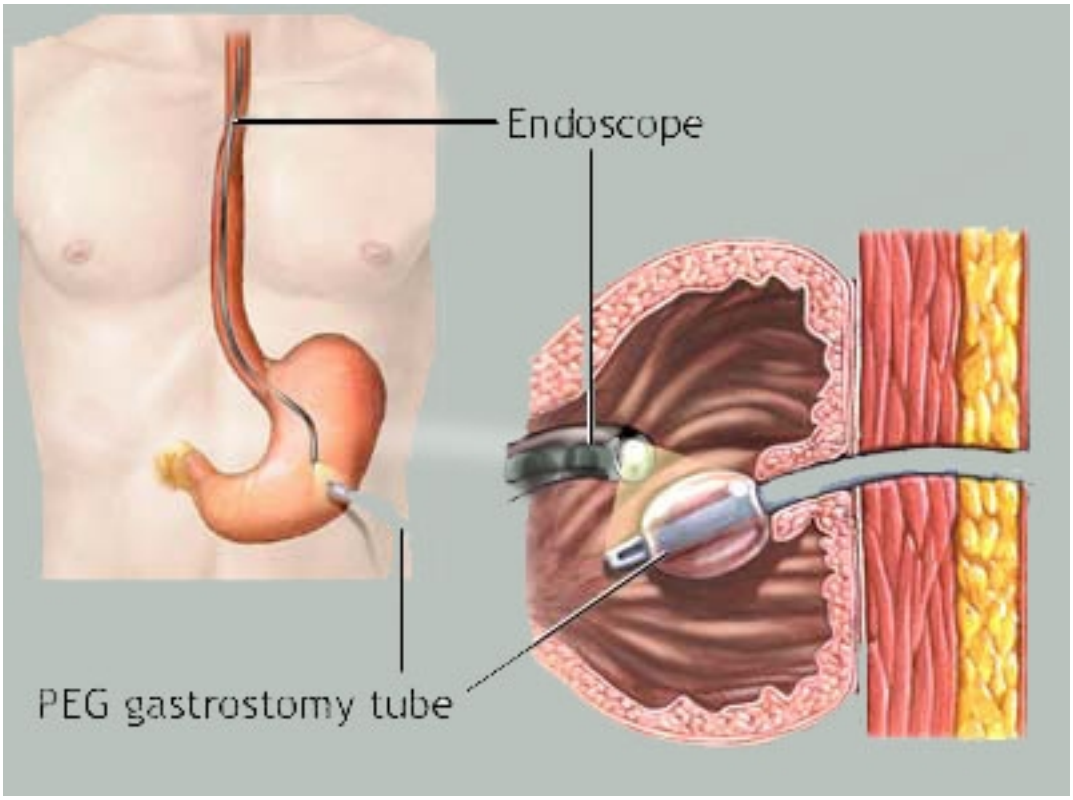
The "Pull" Technique

The pull technique, first introduced by Ponsky and Gauderer, is the most commonly used PEG approach. Adult patients are kept nil per os for at least 8 hours before the procedure. They are supine when abdominal landmarks are identified. The insertion site—in the left upper quadrant immediately below the costal margin avoids injury to the liver and transverse colon. After conscious sedation and topical anesthesia of the pharynx, the gastroscope is passed into the stomach. The esophagus, stomach, and duodenum are examined to rule out any abnormal conditions that would contraindicate the use of PEG placement.



Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

A).PEG insertion site of the abdominal wall. B) The endoscopist observes one-to-one indentation of the stomach by the assistant's finger applied to the anterior abdominal wall.



Under normal conditions, the anterior abdominal wall is transilluminated by the endoscope light. The stomach is insufflated with air and kept distended throughout the procedure. The abdominal wall is cleaned with antiseptic. Local anesthesia is administered at the puncture site, and a 0.5-cm incision is made. A large-bore needle with an inner stylet is passed from outside the body into the stomach. Once the position of the needle is confirmed, the stylet is removed and a string is introduced. A biopsy snare is used to grasp the tip of the needle to remove the string through the patient's mouth. The endoscope is also withdrawn. The feeding tube is attached to the string tip and pulled out through the patient's abdominal wall. The proper position of the tube in the stomach is confirmed by endoscopy. The insufflated air is evacuated and the tube is secured to the skin by a retention disk.

The "Push" Technique

The push and pull techniques are similar. The only difference between them is that with the push approach, a longer feeding tube is used.

Complications of Percutaneous Endoscopic Gastrostomy

PEG has a mortality rate of around 1% and a morbidity rate of around 6%. Complication include feeding tube dislodgement, bleeding, surgical site infection, peritonitis, septicaemia, peri-tubal leakage, gastro-oesophageal aspiration, perforation of the bowel, and the formation of internal fistula. Peri-stomal wound infection, which occurs in as many as 30% of patients, is the most frequently reported complication.

Data indicate that use of a prophylactic antibiotic can reduce the risk of infection at the PEG site.

Surgical Gastrostomy

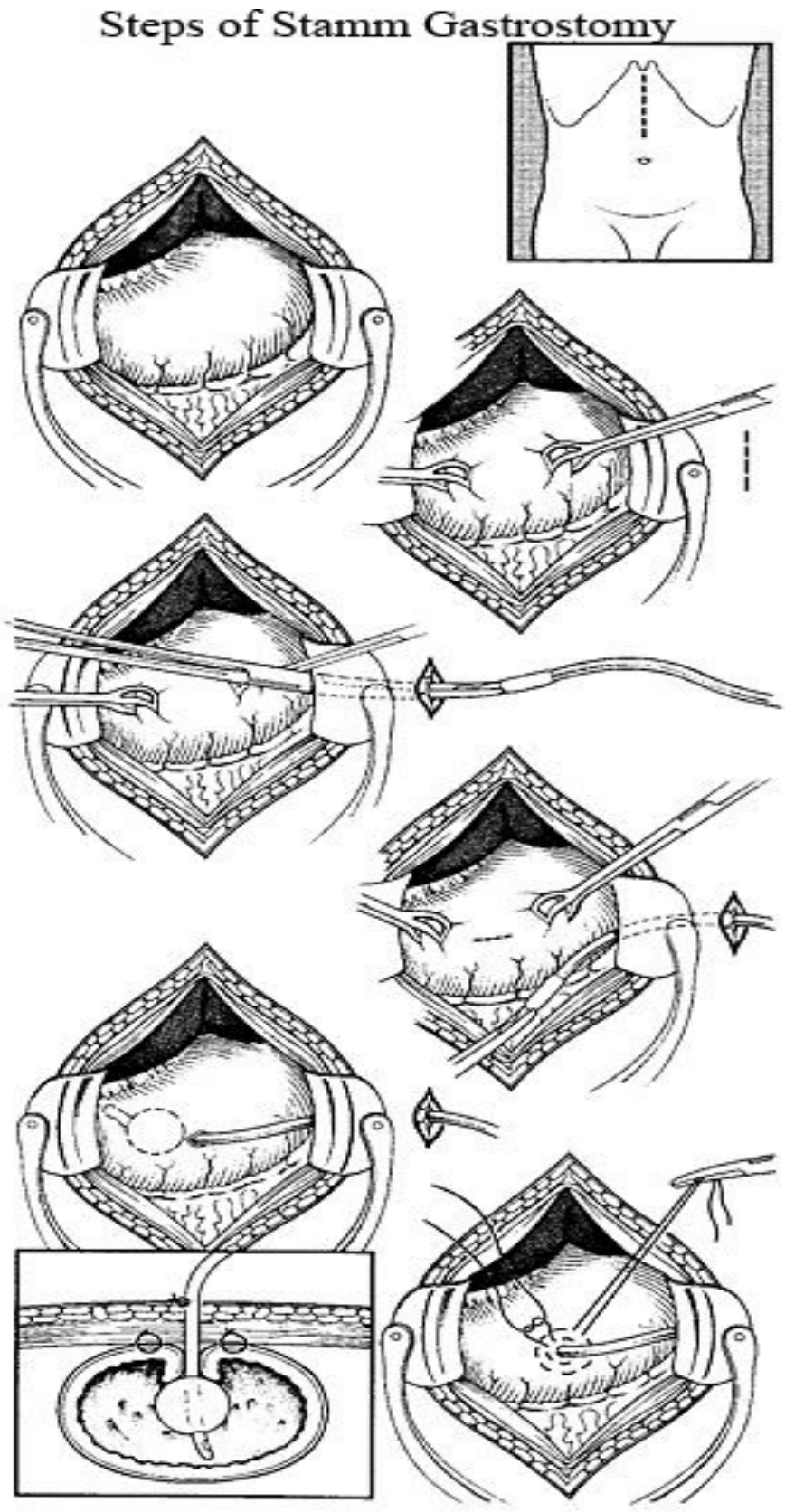
Surgery is indicated when percutaneous placement of gastrostomy tubes is not possible or is contraindicated (e.g., patients with previous abdominal or stomach surgery, esophageal obstruction, or overlying liver or colon). The procedure is also used in some trauma patients and those undergoing abdominal surgery that requires gastric access. The two most commonly performed techniques are Stamm and Witzel gastrostomies.

Stamm Gastrostomy Technique

Stamm gastrostomy is easy to perform. When properly executed, it is associated with minimal morbidity. Modified Stamm gastrostomy can be performed under local anesthesia.

A vertical midline incision is made between the umbilicus and xyphoid process. The peritoneum is entered and the stomach brought into view by gentle retraction of the omentum. Two Babcock clamps are used to grasp the anterior gastric wall near the greater curvature. Two purse-string sutures are placed in the seromuscular layer. A stab wound is made through the left anterior abdominal wall approximately 4 cm lateral to the incision and 2 cm inferior to the costal margin. The gastrostomy tube is passed through the stab wound into the peritoneum cavity.

The stab wound is then made in the center of the purse-string sutures in the stomach wall and the gastrostomy site is opened with a hemostat. Next, the tube is inserted into the stomach through the gastrostomy site and the purse-string sutures are tied. The tube is retracted and apposed to the peritoneum and the stomach wall. The stomach is sutured to the anterior abdominal wall and the tube is sutured to the skin. The midline incision is then closed. The steps are illustratred in the figures below.



Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

Witzel Gastrostomy Technique

The key difference between the Stamm and Witzel gastrostomies is the creation of a tunnel in the abdominal wall to minimize leakage. It is also more difficult to replace tubes using the Witzel technique.

Laparoscopic Gastrostomy

This minimally invasive laparoscopic technique has several potential advantages, including (a) a smaller incision, (b) less postoperative pain, (c) decreased wound complications, and (d) the ability to visualize the entire peritoneal cavity. Laparascopic gastrostomy can be performed using several techniques. Each technique involves peritoneal access, creation of a pneumo-peritoneum, placement of the gastrostomy tube, and fixation of the stomach to the anterior abdominal wall.

Postoperative Care

Before initiation of feeding, the gastrostomy tube is used as a gravity drain for approximately 24 hours postoperatively. If the the drain is less than 500 mL and the bowel functions are normal, the gastrosotomy tube is clamped and 5% dextrose infusion is started via the tube. If the patient tolerates this infusion, feeding can be started, usually on the 1st or 2nd postoperative day.

Isotonic formulas are recommended for initial feeding. Volume can be increased as the patient tolerates. In critically ill patients, continuous rather than intermittent feedings should be used to decrease risk of aspiration and gastric

distention and reduce metabolic complications. Gastric residual volume should be assessed 4th hourly until it decreases to less than 50ml.

There is no consensus on acceptable volumes of gastric residual. In general, feeding should be withheld if residual volume exceeds 200 mL with nasogastric tubes or 100 mL with gastrostomy tubes on two consecutive assessments. Patients with persistently elevated gastric residuals may require jejunal feeding.

Complications

Major complications occur after open gastrostomy in approximately 7% to 15% of patients. Perioperative problems include:

1. Bleeding at the gastrostomy site;
2. Injury to other abdominal structures during tube placement.

Postoperative complications include:

1. Surgical site infections/ wound dehiscence;
2. Intraperitoneal and peristomal leakage;
3. Ileus.

Long-term complications include tube dislodgement, stomach obstruction, and clogging. Dislodgement of new gastrostomy tubes could result in intraperitoneal leakage of intestinal content and peritonitis. Because tracts can close quickly, replacement of tubes with well-formed tracts should be done immediately. Clogging is usually caused by thick formula or precipitated medications. Regular flushing can avert this complication.

Jejunostomy

Percutaneous Jejunostomy

Percutaneous jejunostomy can be performed using radiologic (fluoroscopic, ultrasonographic, tomographic) or endoscopic guidance. Although this technique is relatively safe, it lacks wide acceptance. This is probably the result of technical difficulties, a high probability of puncture of the mobile and compliant jejunum, and difficulty in maintaining the position of the jejunum during catheter insertion. Common complications include peri-tubal leakage and surfical site infections..

Surgical Jejunostomy

Surgical jejunostomy is indicated when the stomach cannot be used or the patient is at high risk for aspiration. It is many a time used as an add-on to a major abdominal surgery with anticipated prolonged fasting. Examples include hepatic, pancreatic, esophageal, and gastric operations. Other indications for jejunostomy include moderate to severe malnourishment, patients who are to undergo chemotherapy or radiotherapy in the postoperative period, intra-abdominal organ transplant, sepsis, and extensive intra-abdominal trauma.

Jejunostomy Techniques

There following are the currently practiced techniques for insertion jejunostomy tube surgically:

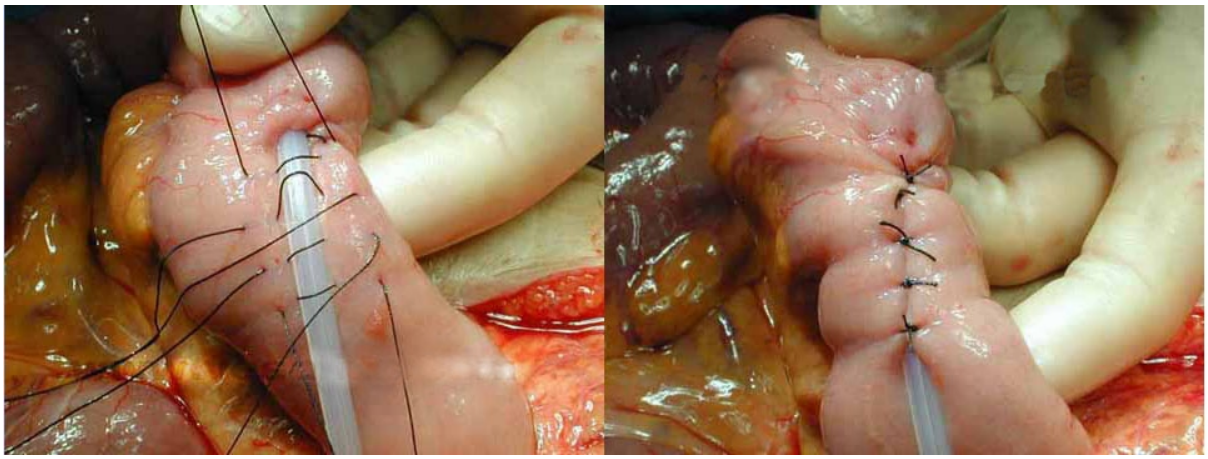
1. Longitudinal Witzel;

2. Transverse Witzel;
3. Open gastrojejunostomy;
4. Needle catheter;
5. Laparoscopy.

Despite frequent complications (i.e., tube dislodgement and obstruction caused by narrowing of the intestinal lumen), Witzel procedures are popular methods for placing jejunostomy tubes. They entail the creation of a serosal tunnel on the antimesenteric border of the jejunum.

Longitudinal Witzel Technique

Longitudinal Witzeljejunostomy uses a segment of jejunum that is 20 cm distal to the ligament of Treitz. The site allows simple apposition of the jejunum to the anterior abdominal wall. A small stab wound is made on the antimesenteric border of the jejunum and a rubber tube is inserted distally for approximately 20 cm. A purse-string suture is placed around the tube at the jejunostomy site. A seromuscular incision is made proximally from the tube approximately 5 cm. The jejunostomy tube is placed in the seromuscular tunnel. The tunnel is closed with interrupted sutures and the tube is brought out through the anterior abdominal wall via a separated incision. The jejunum is then sutured to the abdominal wall.



Longitudinal Witzel Technique

Tranverse Witzel Technique

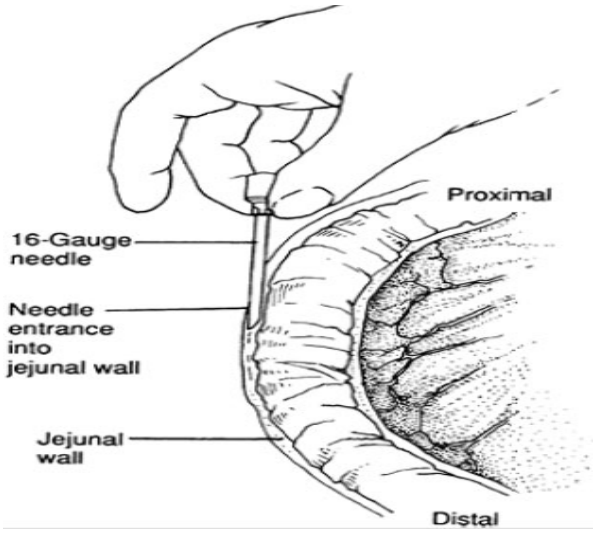
The transverse Witzel technique substitutes a standard French rubber catheter with a T-tube that is transversely sutured to the mesenteric border. The transverse Witzeltechnique appears to decrease dislodgement, compared with its longitudinal counterpart.

Needle Catheter Technique

The needle catheter technique is a simple and safe method for jejunostomy placement. The technique is more advantageous, including fewer complications compared with Witzel methods, and has the ability to infuse additional fluids, electrolytes, and drugs that might otherwise require a central line. Prior to the introduction of the needle tip into the lumen, a 14-gauge needle that is 7 cm long 14G needle is inserted intramurally into the wall of the proximal jejunum parallel to the long axis for approximately 5 cm . A 16G catheter with a stylet is threaded through

the needle into the intestinal lumen for approximately 25 cm. The stylet and the needle are then withdrawn and the catheter is secured with a purse-string suture.

A separate needle is inserted at approximately at a 45-degree angle into the greater sac through the anterior abdominal wall, and the opposite end of the catheter is threaded backward to exit the abdominal wall. The needle is removed, the catheter is secured to the skin, and the jejunum is sutured to the anterior abdominal wall. The catheter should then be flushed to confirm that there are no occlusions or kinks.

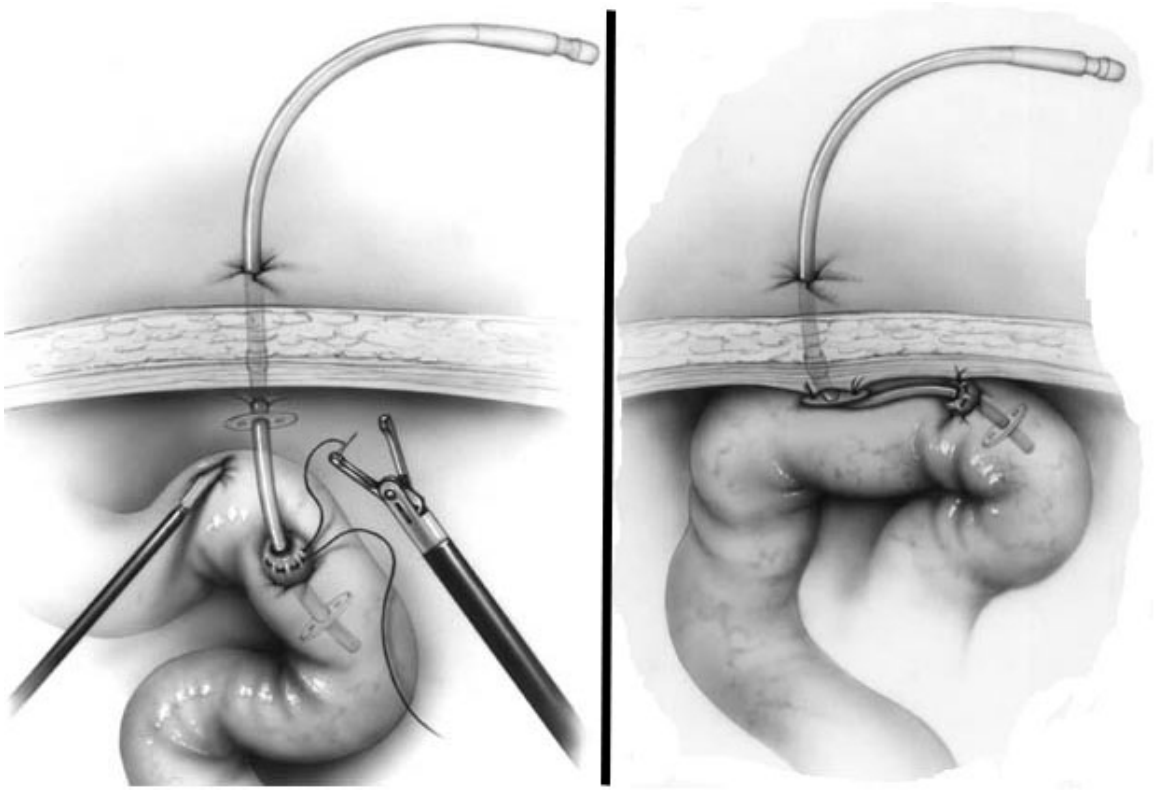


Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

Laparoscopic Jejunostomy

Indications for laparoscopic jejunostomy are similar to those for jejunostomy. The procedure is usually performed in patients undergoing other procedures (laparoscopically or under general anesthesia) that require nutrition access. It is also indicated when the stomach is not accessible. Several techniques for performing laparoscopic jejunostomy have been described.

These include laparoscopic-assisted jejunostomy, needle catheterization jejunostomy, intracorporeal suturing techniques, andtransabdominal fixation using sutures or T-fasteners. In general, laparoscopic placement of a jejunostomy tube is more difficult and time-consuming than a gastrostomy. It is also more expensive. In one study, laparoscopic jejunostomy was found to be almost three times more costly than PEG, and two times more costly than surgical jejunostomy. The potential advantages of laparoscopic jejunostomy include reduction in postoperative pain, shorter rehabilitation time, and decreased risk of wound infection.



Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

Jejunostomy Complications

Complications from jejunostomy are similar to those from gastrostomy. Problems reported in Witzel procedures include obstruction, intraperitoneal leakage, infection, and tube dislodgement. Two percent of complications are associated with the needle catheter method. These include withdrawal and obstruction of the catheter, pneumatosisintestinalis, abdominal wall infection, subcutaneous abscess, intestinal occlusion and volvulus

Combined Gastrostomy–Jejunostomy Tube

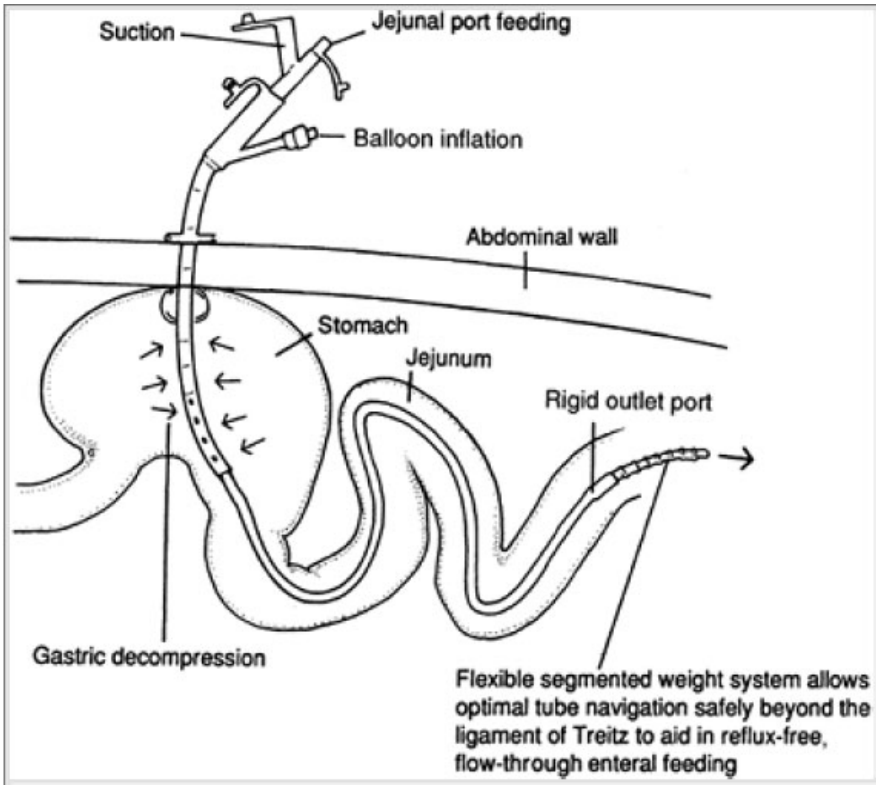
The combined gastrostomy–jejunostomy tube provides two exclusive ports for jejunum and stomach. Simultaneous feeding via the jejunal port and decompression of the stomach via the gastric port, decreases the risk of aspiration. As the jejunum is not opened, the procedure eliminates such complications as peritoneal leak, intestinal volvulus, and luminal narrowing.

Gastrostomy-Jejunostomy Techniques

There are several ways to place a gastrostomy-jejunostomy tube. The gastric port is a wide bore tube placed in the stomach as in a standard gastrostomy. The jejuna port is a smaller bore tube that is manoeuvred via the gastrostomy tube and then advanced in to the jejunum.

In open surgical placement, the jejunal tube can be manipulated through the pylorus and then manually advanced and palpated when it has reached the jejunum. In percutaneous technique, the jejunal tube is introduced via a PEG; it is then grasped

with biopsy forceps, and advanced with an endoscope inserted in the stomach. Once in the jejunum, the endoscope is retracted, leaving the jejunal tube in-situ. Jejunal tubes can also be positioned using fluoroscopy.



A Gastrostomy-jejunostomy catheter. G–J catheter with simultaneous gastric decompression and jejunal feeding. The separate portal of entry allows for jejunostomy tube replacement independent of the gastric tube.

Source: FISCHER, J. E., & BLAND, K. I. (2007). Mastery of surgery

The different routes of enteral feeding are summarized in the following table:

Enteral Nutrition Feeding Routes

ROUTE	SUITABILITY	INSERTION METHOD, CONFIRMATION	ADVANTAGES	DISADVANTAGES
Nasogastric	Short term – functional GI tract	Blind at bedside; Fluoroscopy guided	Easy to insert, replace; can monitor gastric pH and residual volume; bolus feeding	Aspiration risk, misplacement complications, sinusitis, epistaxis, nasal necrosis, esophageal strictures, erosive esophagitis
Nasoduodenal, nasojujenal	Short term – functional GI tract but poor gastric emptying, reflux, aspiration risk; commence feed only when volume-resuscitated and hemodynamically stable	Blind at bedside; fluoroscopy guided, endoscopy guided	Reduced aspiration risk, some tubes enable decompression of stomach while feeding into jejunum	Easily clogged or displaced, aspiration risk, misplacement complications, displacement and reflux into stomach, sinusitis, epistaxis, nasal necrosis; required continuous infusion; cannot check gastric residuals except with specialized gastric port
Gastronomy	Long term – good gastric emptying; avoid if significant reflux or aspiration problem	Surgical, percutaneous, endoscopic, radiologic	Bolus feeding; large-bore tube less likely to block	Procedure risks include bleeding, perforation, aspiration risk, dislodgement with peritoneal contamination, wound site infection, granulation
Jejunostomy	Long term – functional GI tract but poor gastric emptying, reflux, aspiration risk, gastroparesis	Surgical, percutaneous, endoscopic, radiologic	Reduced aspiration risk	Bleeding, infection, perforation, migration, aspiration, dislodgement & leakage into peritoneal cavity, occlusion, pneumatosis, intestinal ischemia or infarction, bowel obstruction; difficult to replace; cannot check residuals; requires continuous infusion

Complications of Enteral Nutrition

There are four categories of EN complications: gastrointestinal, infectious, metabolic, and mechanical.

Gastrointestinal

The common GI complications due to the enterally feeding the patients are as follows:

1. Abdominal bloating;
2. Diarrhea/constipation;
3. Nausea, and vomiting.

Abdominal distention and cramps can result from delayed gastric emptying, intestinal obstruction, or fermentation of the diet. Diarrhoea occurs in 10% to 20% of patients; however, other causes of diarrhoea (e.g., Clostridium difficile colitis) should be considered. Diarrhoea may result from an overly rapid increase in the volume of hyperosmolar tube feedings, medications (e.g., metoclopramide), a high-fat diet, or the presence of components not tolerated by the patient (e.g., lactose). If other causes of diarrhea can be excluded, the volume or concentration of tube feedings should be decreased. Decreased infusion rate, followed by gradual increases, may also help intestinal adaptation. Antidiarrhoeal agents such as loperamide should be reserved for patients with severe diarrhoea who have had infectious etiologies excluded. . In some patients who do not receive broad-spectrum antibiotics, supplementation of formula with fibre may be helpful.. If diarrhoea persists, TPN may be necessary.

Infectious

Tracheo-bronchial aspiration of tube feeds may occur with patients who are fed into the stomach or proximal small intestine and can lead to major morbidity. Patients at particular risk are those with central nervous system abnormalities and those who are sedated. Precautions include frequent assessment of gastric residuals as well as head of bed elevation. Small bowel feedings may decrease the incidence of aspiration (neuropathy, and myocardial infarction). The presence of a NG tube across the lower oesophageal sphincter can impair its function and can cause gastro-oesophageal reflux. Anti-reflux medications are not of much help in these situations where there is a mechanical hindrance to the normal function of the sphincter.

Metabolic

Metabolic complications associated with EN include fluid and electrolyte disorders, hyperglycemia, vitamin and trace element deficiencies, and refeeding syndrome. Careful monitoring—especially in patients with renal, hepatic or cardiac insufficiencies can reduce these complications.

Refeeding syndrome, a complication of aggressive nutrition support, can be dangerous if not treated promptly. High-risk patients have marasmus with prolonged starvation. Rapid reintroduction of a large amount of glucose in such patients can lead to hypomagnesemia, hypophosphatemia, hypokalemia, and fluid retention. Hypophosphatemia can result in neuromuscular, respiratory, and cardiac dysfunction. Refeeding problems are avoided by very low rate feeding, and treatment of any electrolyte imbalance, particularly in severely malnourished patients. Hyperglycemia

occurs in 10% to 30% of tube-fed patients. It is usually associated with high carbohydrate formulas. The condition is more likely to occur in patients with insulin resistance related to their illness or in patients with undiagnosed diabetes or in patients with sepsis. Treatment includes switching to low-carbohydrate formulas. Administration of insulin or antidiabetic agents may be necessary. A sliding scale insulin protocol along with long-acting agents should be used to treat hyperglycemia in tube-fed patients

Mechanical

Mechanical complications are a consequence of feeding tube insertion and placement. Nasoenteral tubes may cause nasopharyngeal discomfort, sore throat, thirst, swallowing difficulty, and hoarseness. Local pressure from the tubes may result in nasopharyngeal erosion, sinusitis, otitis media, oesophagitis, oesophageal ulcer, gastro-oesophageal reflux, trachea-oesophageal fistulas, and rupture of oesophageal varices. Such damage is rare with fine-bore tubes. Tubes should be replaced every 4 to 6 weeks, with insertion in the alternate nostril. In patients with acute variceal bleeding, NG tubes must avoided for atleast 3 days. Clogging can usually be prevented by careful routine flushing of the feeding tube. Instillation of carbonated soda, cranberry juice, or pancreatic enzyme replacement is sometimes useful for unclogging feeding tubes.

PATIENT EVALUATION

Nutritional Assessment

The main goals of nutritional assessment are (a) to identify patients who have, or are at risk for, protein-energy malnutrition or nutrient deficiency, and (b) to assess their risks for developing nutrition-related complications.

History and Physical Examination

The most important parts of nutritional assessment are the patient history and physical examination. Measurement of body weight and height can be used to compare actual and ideal body weight. Body mass index (BMI = weight [kg]/ height [m2]) can be used to estimate body fat. Unintentional weight loss greater than 10% within the previous 6 months indicates malnutrition and is associated with poor clinical outcome.

Biochemical Tests

Biochemical tests include those for serum albumin, prealbumin, retinol-binding protein, transferrin, and creatinine.

Serum Albumin

Low serum levels of albumin have been shown to correlate with increased morbidity and mortality in hospitalized patients. However, the biomarker has relatively poor sensitivity and specificity for protein malnutrition, and low levels of serum albumin can be seen in several conditions (e.g., inflammatory, gastrointestinal,

cardiac, kidney, and liver diseases). Plasma albumin is usually unaffected by nutrition intake and does not normalize in stressed patients until inflammatory stress is resolved.

Prealbumin and Retinal-Binding Protein

Prealbuminis a transport protein for thyroid hormones and a binding protein for retinol-binding protein. Its half-life is 2 to 3 days. Energy and protein restrictions lower prealbumin levels, and refeeding restores them. Infection, liver, and kidney failure may affect plasma concentrations of prealbumin.

Transferrin

Transferrin has a shorter half-life than albumin, but the serum levels depend on a patient's iron store. Transferrin helps identify those who are most likely to develop malnutrition and require aggressive and closely monitored medical nutrition therapy.

Creatinine

Twenty-four hour urinary excretion of creatinine can provide an index of lean body mass. Accuracy of the index requires consumption of a meat-free diet and effective and complete urine collection.

Clinical Test

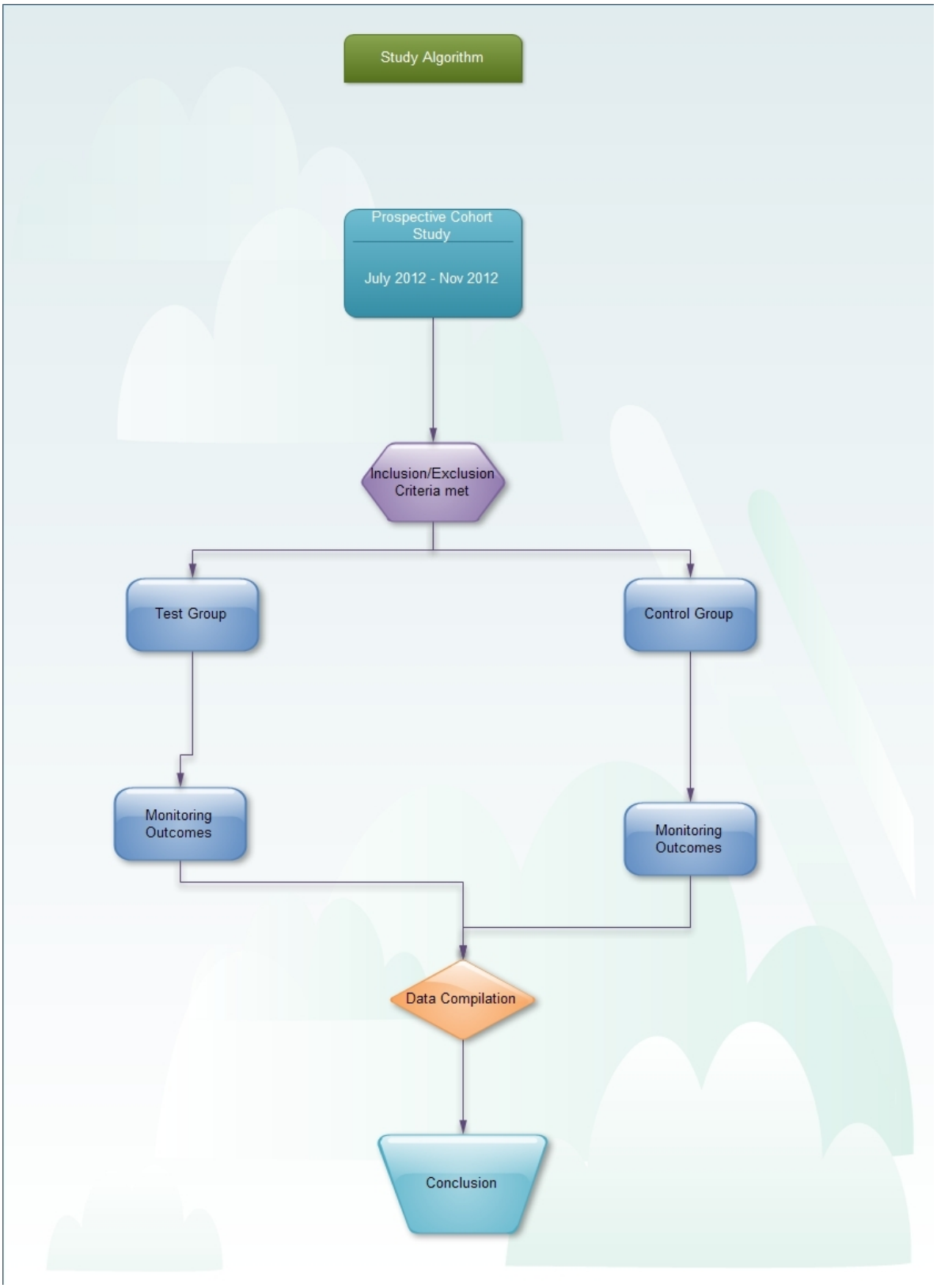
Subjective Global Assessment

Subjective global assessment is a clinical method for determining nutritional status.

METHODOLOGY OF STUDY

The following study was conducted in Kilpauk medical college and hospital. It is a prospective cohort interventional study, the source of the study being patients admitted in general surgery and surgical gastroenterology wards for either gastrointestinal surgeries or acute pancreatitis. The period of longitudinal observation was from July 2012 to November 2012. Inclusion and exclusion criteria were drawn up and only those patients satisfying both these criteria were included in the study. Patients admitted in my unit for GIT surgeries or acute pancreatitis constituted the test group while patients, while patients admitted in other units for similar disease processes constituted the control group. The sample size of the study was fixed at 100, the breakdown of which is as follows:

- **Test group (TG) – Patients were pooled from my unit (25 patients undergoing GIT surgeries + 25 patients diagnosed with acute pancreatitis);**
- **Control group (CG) – Patients were pooled from neighbouring units (25 patients undergoing GIT surgeries + 25 patients diagnosed with acute pancreatitis).**


Study Algorithm
Prospective Cohort Study
July 2012 - Nov 2012
Inclusion/Exclusion Criteria met
Test Group
Control Group
Monitoring Outcomes
Monitoring Outcomes
Data Compilation
Conclusion

Criteria for Patients undergoing gastrointestinal surgeries

Inclusion Criteria	Exclusion Criteria
• Patients undergoing surgeries of the gastrointestinal system • Age 12 years or older • Informed written consent	• Relaparotomies • Patients with renal failure

Criteria for Patients with Acute Pancreatitis

Inclusion Criteria	Exclusion Criteria
• Diagnosis of Acute Pancreatitis • Age 12 years or older • Informed written consent	• >48 hours after onset of symptoms • Severe Pancreatitis • Chronic Pancreatitis • Post ERCP Pancreatitis • Pregnancy • Malignancy • Pantients with Renal failure

DATA COLLECTION

Ours is a tertiary health centre that offers free health care to the population. The general surgery and the surgical gastroenterology department cater to patients with surgical pathology; both emergent and elective scenarios.

Patients admitted in the general surgery and SGE wards who fulfilled both the inclusion and exclusion criteria were included in the study. Primarily, all relevant findings, both subjectively and objectively, that were thought to have influenced the recovery of the patient were recorded. A note to be made at this juncture is that only investigations that were available in our hospital could be included; a few of the investigations that would throw a better light on the recovery process could not be done because of their non- availability. But as with any government institution, financial constraints go hand in gloves.

A proforma was prepared to record the findings. The patients and the attenders were explained about the study and informed written consent was sought from all of them.

In the test group with patients undergoing GIT surgeries, enteral feeds were started withing 24 hours of the procedure. On the first post operativeday, nasoenteric feeding was initiated with clear liquids at the rate of 30ml/hr. Around 200 ml of this feed was continued. Clear liquids consisted of tender coconut water, barley water. Subsequently the proposed diet was started in these patients at the rate of 30 ml/hr. Around 500 ml of this diet was fed intermittently. The diet proposed by the nutrionist

was that of a "Rice Conjee". This was found to have many advantages. It was accepted by all the patients, it was easy to prepare, and it was cost effective. Moreover, the inherent scarcity of fatin this preparation, makes it ideal to be used among patients with acute pancreatitis. The nutritional value of the diet is enumerated in the table below.

Rice Conjee – 500 mL

Ingredients

1. Rice: 25g;
2. Roasted Bengal gram: 20g;
3. Sago: 20g;
4. Ragi: 25g;
5. Jaggery: 5 g;
6. Milk: 200mL

	Quantity	Energy	Protein	CHO	Fat	Iron	Calcium	Fibre	Sodium	Potassium
Rice	25 g	87.25	2.1	19.35	0.15	0.80	2.20	-	-	-
Roasted Bengal goan	20 g	73.80	4.5	11.62	1.04	1.90	11.6	0.20	-	-
Sago	20 g	70.20	0.04	17.42	0.04	0.26	2.00	-	-	-
Ragi	25 g	80.00	1.75	1.75	0.25	0.75	85.00	0.95	2.75	102.00
Jaggery	5 g	20.00	0.02	4.75	0.005	0.20	120.00	-	73.00	140.00
Milk	200 mL	134.00	6.00	8.00	8.00	0.40	240.00	-	144.00	45.40
Total		**465 kcal**	**14 g**	**63 g**	**9 g**	**4 g**	**460 g**	**1.1 g**	**220 mg**	**290 mg**

Starting from the second post operative day, in patients with nasogastric tube and who had volitional control, the tube was removed and patients were fed orally. The same diet was continued, albeit at a rate of 60ml/hr. Around 1500 ml of diet was given on the second day. Subsequently the patients were switched on to an acute special diet, the details of which are given in the table.

In the patients with acute pancreatitis within the test group, a similar protocol was followed. For the first two days nasogastric feeding was initiated with the "Rice conjee" diet and subsequently switched over to oral feeds; at first with the same diet and later with an acute special diet.

In the control subjects, oral/nasoenteric feeding was initiated, after the initial period of postoperative ileus in the post op patients, or after the period of paralytic ileus in the case of patients with acute pancreatitis. The resolution of the ileus was defined as the return of bowel movements without abdominal distension or vomiting. This was ascertained both subjectively (Passage of flatus) and objectively (return of bowel sounds on auscultation).

A note to be made here is that in all the test group subjects, adequate hydration was maintained with intravenous fluids after subtracting the fluid intake through the diet; while in the control subjects, fluids were given based on the weight of the patient.

For the post operative patients, examination and dressing of the surgical wound was done for the first time on the 2nd post operative day. Sutures if any were removed by the 10th POD. A single dose of antibiotic was administered for the elective cases 30 minutes before the time of induction, while a course of antibiotics were given for the emergency cases, as most of them had frank peritonitis. No antibiotics were given for the patients with acute pancreatitis.

Patients recovery was assessed by clinical symptoms, physical examination and the certain core investigations imperative for the study. They are as follows:

1. Blood Sugar (Daily);
2. Blood Urea/ Serum Creatinine (Daily);
3. Serum Albumin (Day of admission, 5th POD and day of discharge);
4. Liver Function Tests (Day of admission, 5th POD and day of discharge);
5. Ultrasonogram of the abdomen (Day of admission, 5th POD and day of discharge).

The other parameters necessary for the study were recorded by filling up the following proforma and checklist:

Name of Patient											
Age											
Sex	Male □			Female □							
IP No.											
Route of Nutrition											
Weight (in Kg)	**On Admission**			**On 5th post op day/admission day**				**On day of discharge**			
Intake (in mL)	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 5**	**Day 6**	**Day 7**	**Day 8**	**Day 9**	**Day 10**	**Day of discharge**
Output (in mL)	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 5**	**Day 6**	**Day 7**	**Day 8**	**Day 9**	**Day 10**	**Day of discharge**
Blood Sugar (mg/dL)	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 5**	**Day 6**	**Day 7**	**Day 8**	**Day 9**	**Day 10**	**Day of discharge**
Urea/Cretinine											
Serum/Albumin	**On Admission**			**On 5th post op day/admission day**				**On day of discharge**			
Serum Alkaline po4	**On Admission**			**On 5th post op day/admission day**				**On day of discharge**			

Day of Passage of Flatus/Onset of Bowel												
Symptoms of	**1. Nausea** □											
	2. Vomiting □											
	3. Severe Diarrhea □											
	4. Shortness of Breath □											
Signs of SSI (if yes, on which day)												
Signs of Anastomotic Dehiscence (if yes, on which day)												
Signs of Aspiration/Pneumonia												
Length of Hospital Stay												
Subjective Feeling of Patient												

Checklist

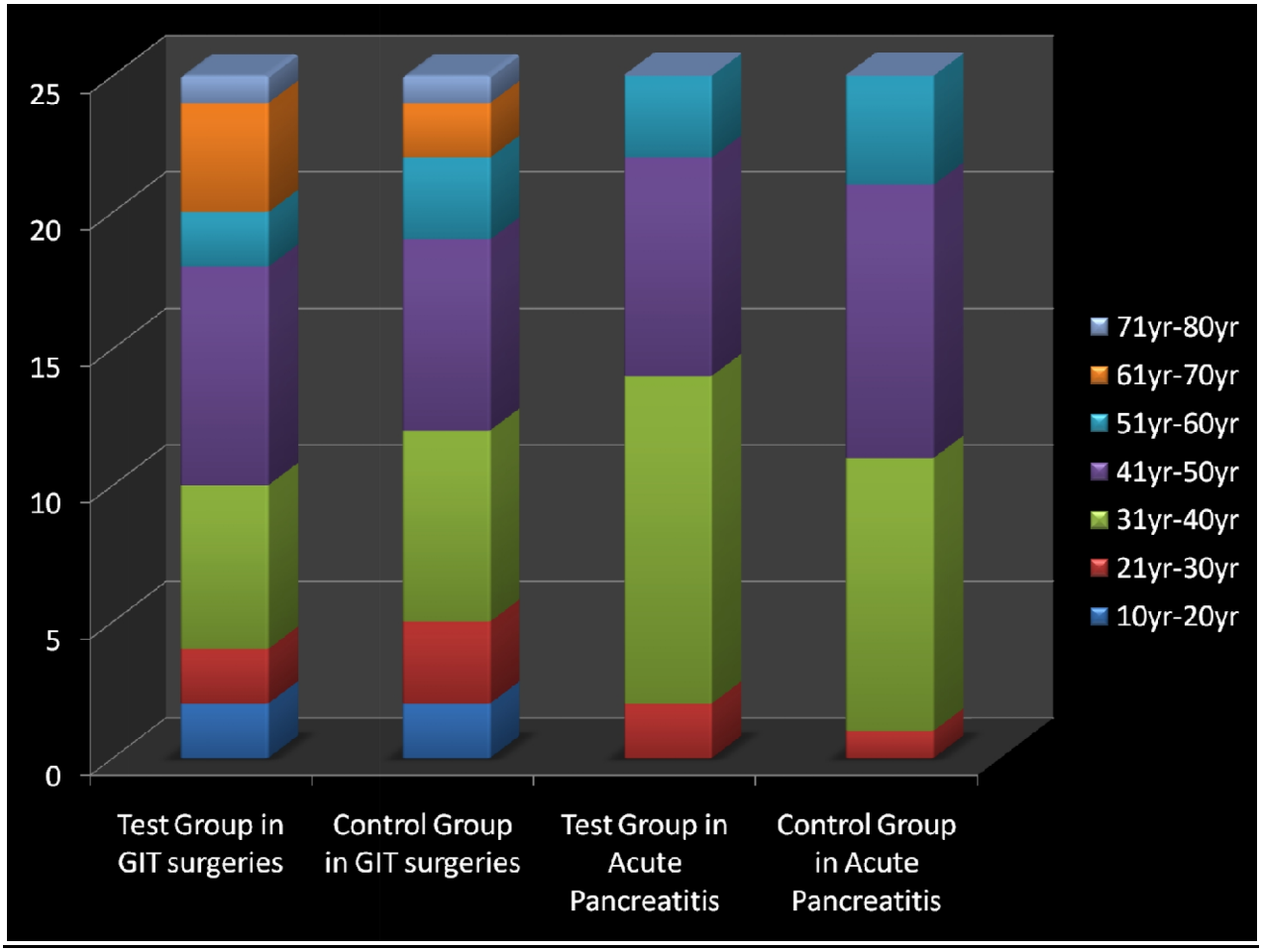
1.	Route of nutrition	□
2.	Routinely flush the tube if any	□
3.	Weigh patient and record on chart on day 1, day 5 ,day 7 and day of discharge	□
4.	Record intake and output daily	□
5.	Record temperature, pulse rate, respiratory rate 2nd hourly	□
6.	Record number, volume, and consistency of bowel movement	□
7.	Obtain complete blood count twice weekly and renal function test everyday	□
8.	Observe for nausea, vomiting, severe diarrhea, or shortness of breath	□
9.	Observe for SSI, Anastomotic dehiscence	□

DATA EXTRACTION & STATISTICAL ANALYSIS

All the relevant data were collected and analyzed using SPSS (Statistical Package for Social Sciences) V.20. Independent 't' test and chi square test were calculated for analysis of the data. A 'p' value of <0.05 was regarded as a significant test value while 'p' > 0.05 was considered not significant.

Results and Observations:

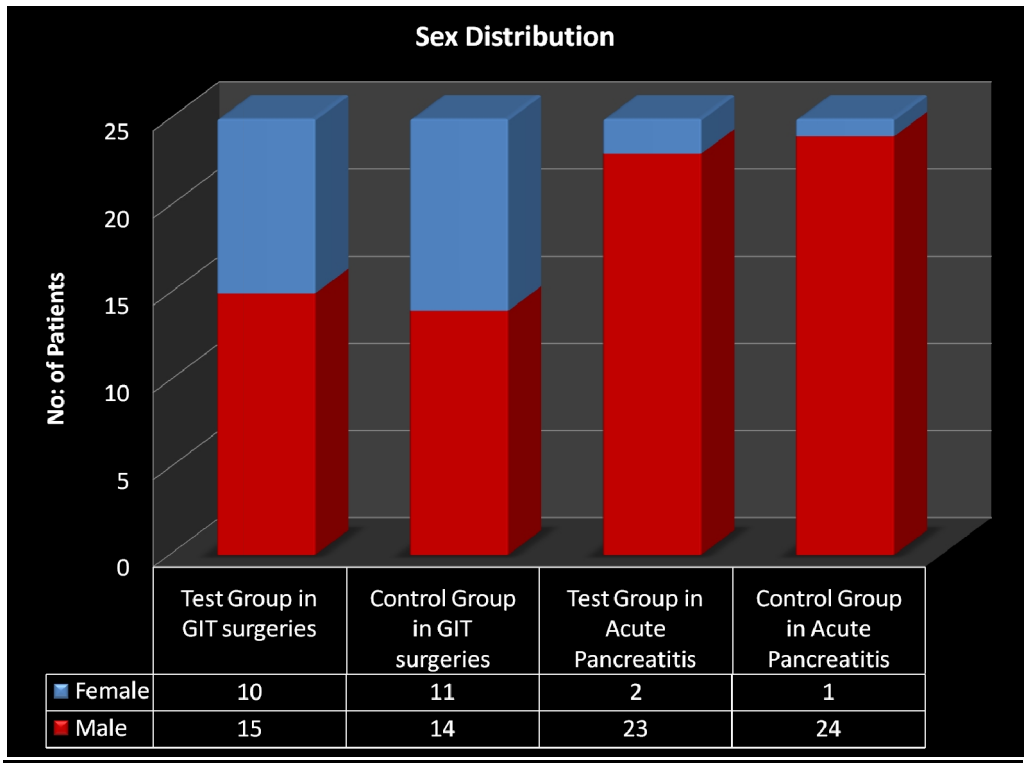
Age and Sex Distribution



Age Groups	Test Group in GIT Surgeries	Group Control in GIT Surgeries	Test Group in Acute Pancreatitis	Control Group in Acute Pancreatitis
10 – 20 years	2	2	0	0
21 – 30 years	2	3	2	1
31 – 40 years	6	7	12	10
41 – 50 years	8	7	8	10
51 – 60 years	2	3	3	4
61 – 70 years	4	2	0	0
71 – 80 years	1	1	0	0

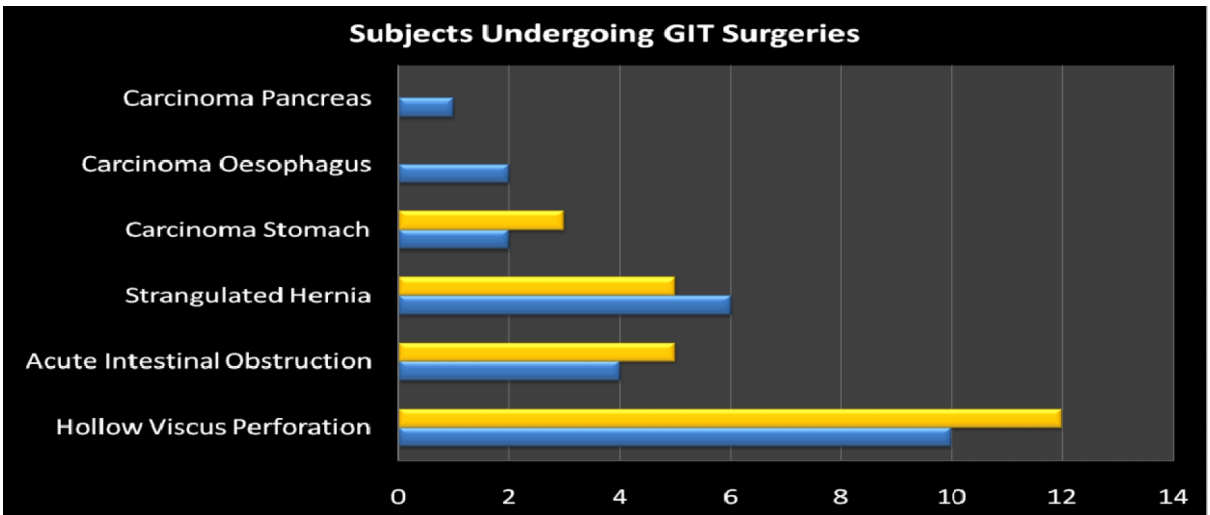
The average age of the patients undergoing GIT surgeries was 33.2 in the test group and 33.4 in the control group. The average age of the patients with acute pancreatitis was 38 in the test group and 37.8 in the control group. As it is evident , the two pairs of groups were similar to one another. The age distribution was also similar among the groups compared. The maximum age of a patient undergoing a GIT surgery in this study was 76, and the minimum age was 12. For patients with acute pancreatitis, the maximum age was 59 and the minimum age was 21.

Sex Distribution



The distribution of patients undergoing GIT surgeries was nearly even among both the sexes. The same cannot be said about patients with acute pancreatitis, which is much more common among males. p=0.02 demonstrating that this was statistically significant.

Breakdown of patients undergoing GIT surgeries

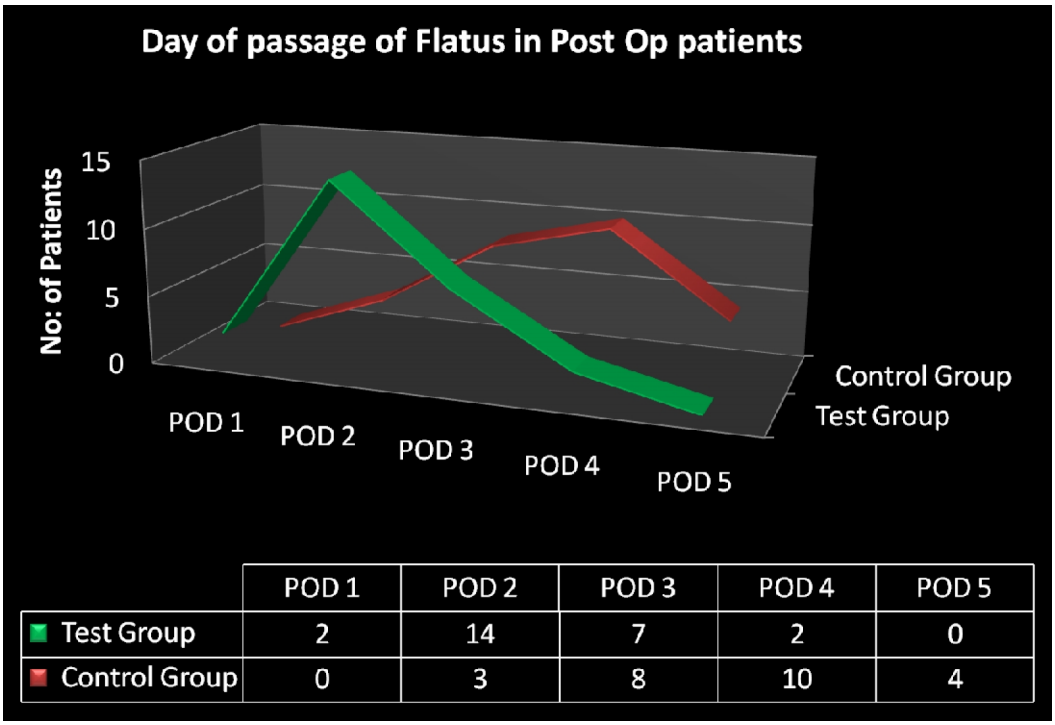


	Hollow Viscus Perforation	Acute Intestinal Obstruction	Strangulated Hernia	Carcinoma Stomach	Carcinoma Oesophagus	Carcinoma Pancreas
Control Group	12	5	5	3	0	0
Test Group	10	4	6	2	2	1

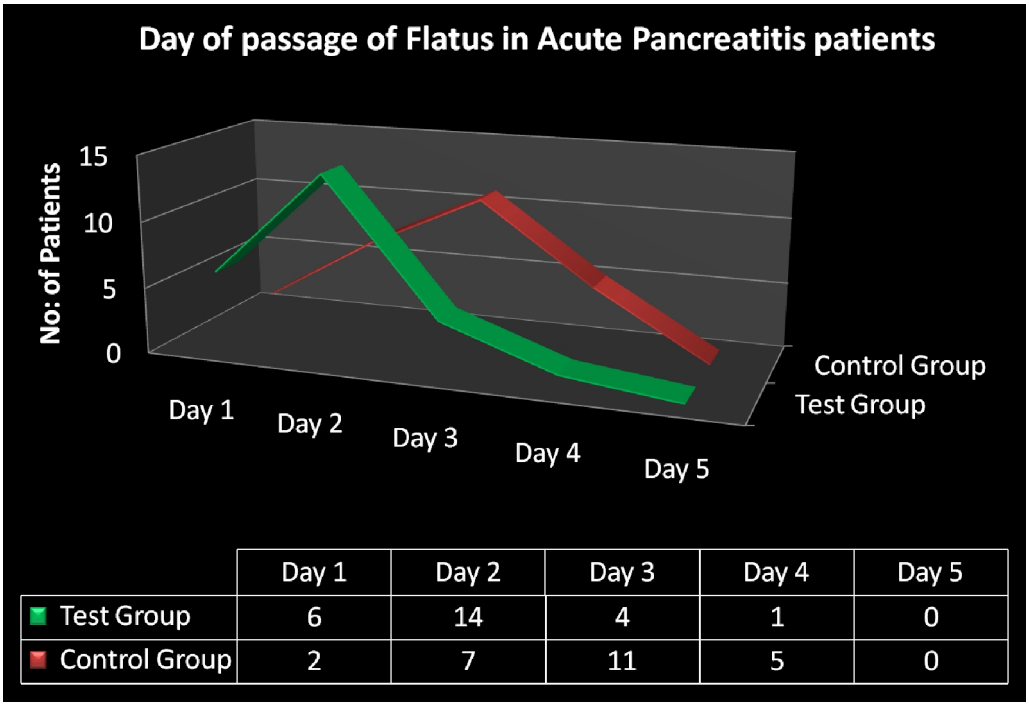
The most common cause for GIT surgeries (besides acute appendicitis, which was excluded from the study) was hollow viscus perforation. Acute intestinal obstruction and and strangulated hernia were the other common causes. Both the test and control groups were comparable in the distribution of the disease process. p = 0.04.

Days to pass flatus

The most common cause for GIT surgeries (besides acute appendicitis, which was excluded from the study) was hollow viscus perforation. Acute intestinal obstruction and and strangulated hernia were the other common causes. Both the test and control groups were comparable in the distribution of the disease process. p = 0.04.



	POD 1	POD 2	POD 3	POD 4	POD 5
Test Group	2	14	7	2	0
Control Group	0	3	8	10	4

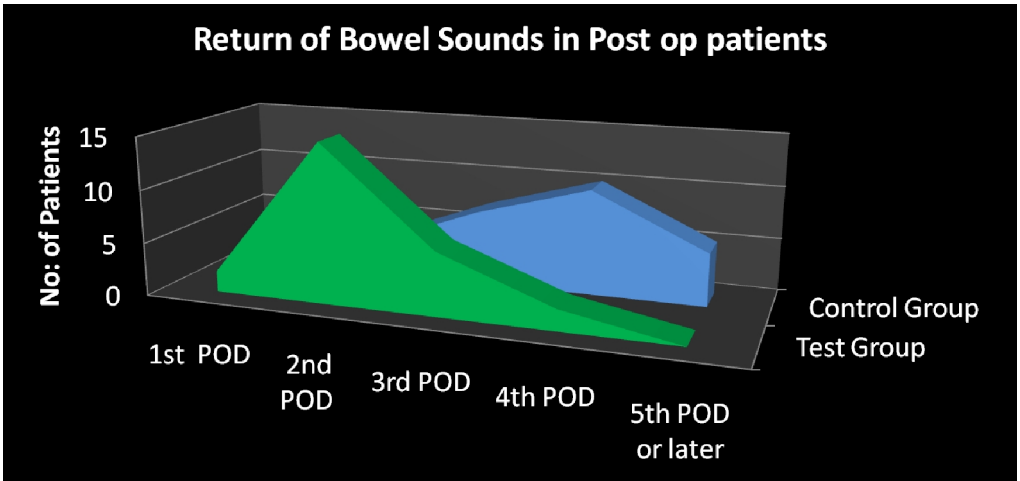


	Day 1	Day 2	Day 3	Day 4	Day 5
Test Group	6	14	4	1	0
Control Group	2	7	11	5	0

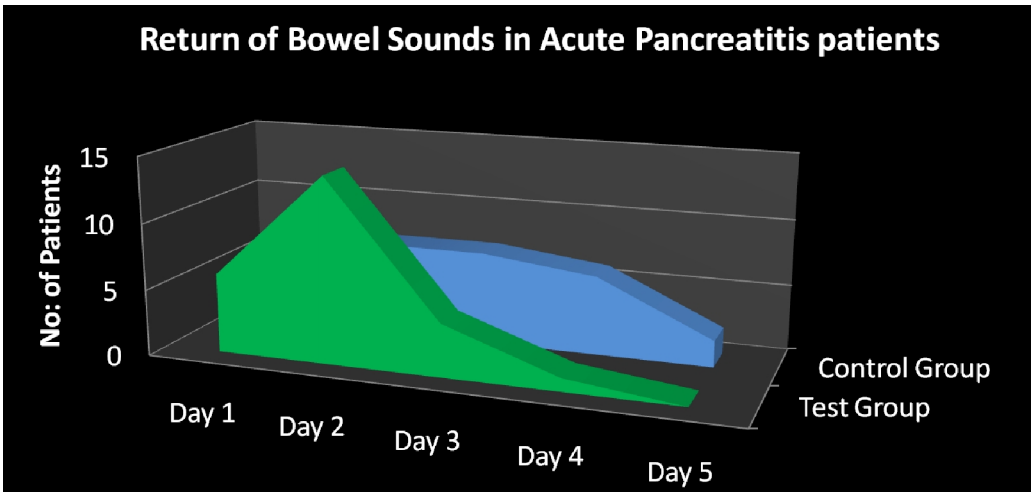
Flatus was passed by the 2nd or 3rd post-operative day among the patients undergoing GIT surgeries in the test group; the mean was 2.2 days (p=0.03). Among the control group, it was between 3rd and 4th Post operative day with a mean of 3 days (0.02).

Among the patients with acute pancreatitis, the mean was 2.1 days in the test group (p=0.01), and 2.9 days in the control group (p=0.02).

Return of Bowel Sounds



	1st POD	2nd POD	3rd POD	4th POD	5th POD or later
Test Group	2	15	6	2	0
Control Group	0	3	7	10	5

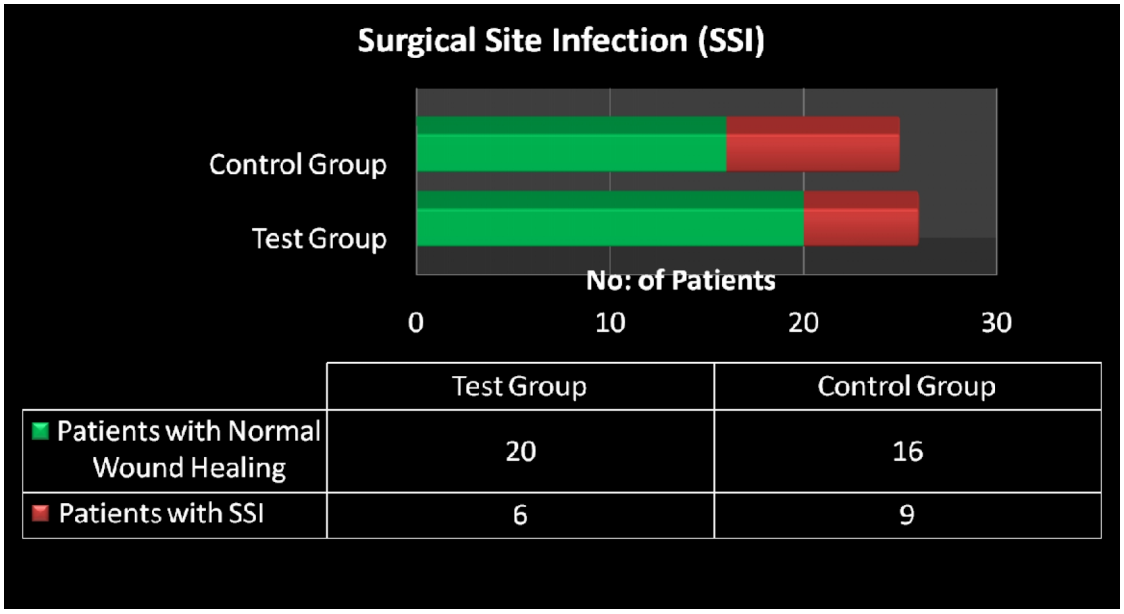


	Day 1	Day 2	Day 3	Day 4	Day 5
Test Group	6	14	4	1	0
Control Group	3	7	7	6	2

Bowel sounds returned between 2nd and 3rd POD among the test group patients undergoing GIT surgeries; mean was 2.3 days (p=0.03). Among the control group, it was between 3rd and 4th POD; mean= 3.4 days (p=0.02).

In patients with acute pancreatitis, mean return of bowel sounds was 1.8 days in the test group (p=0.02); among the control group, mean was 2.9 days (p=0.01).

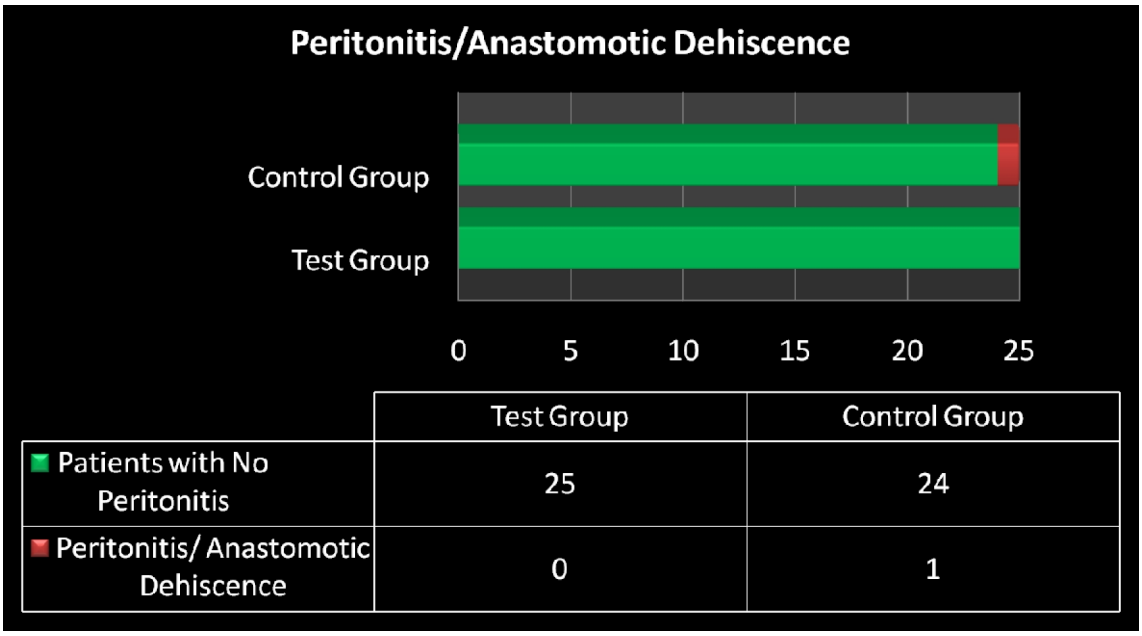
Surgical Site Infection



	Test Group	Control Group
Patients with Normal Wound Healing	20	16
Patients with SSI	6	9

The number of patients with SSI was 6 in the test group and 9 in the control group. Though it did not reach statistical significance (p = 0.08), the infection rate was nevertheless lesser in the test group.

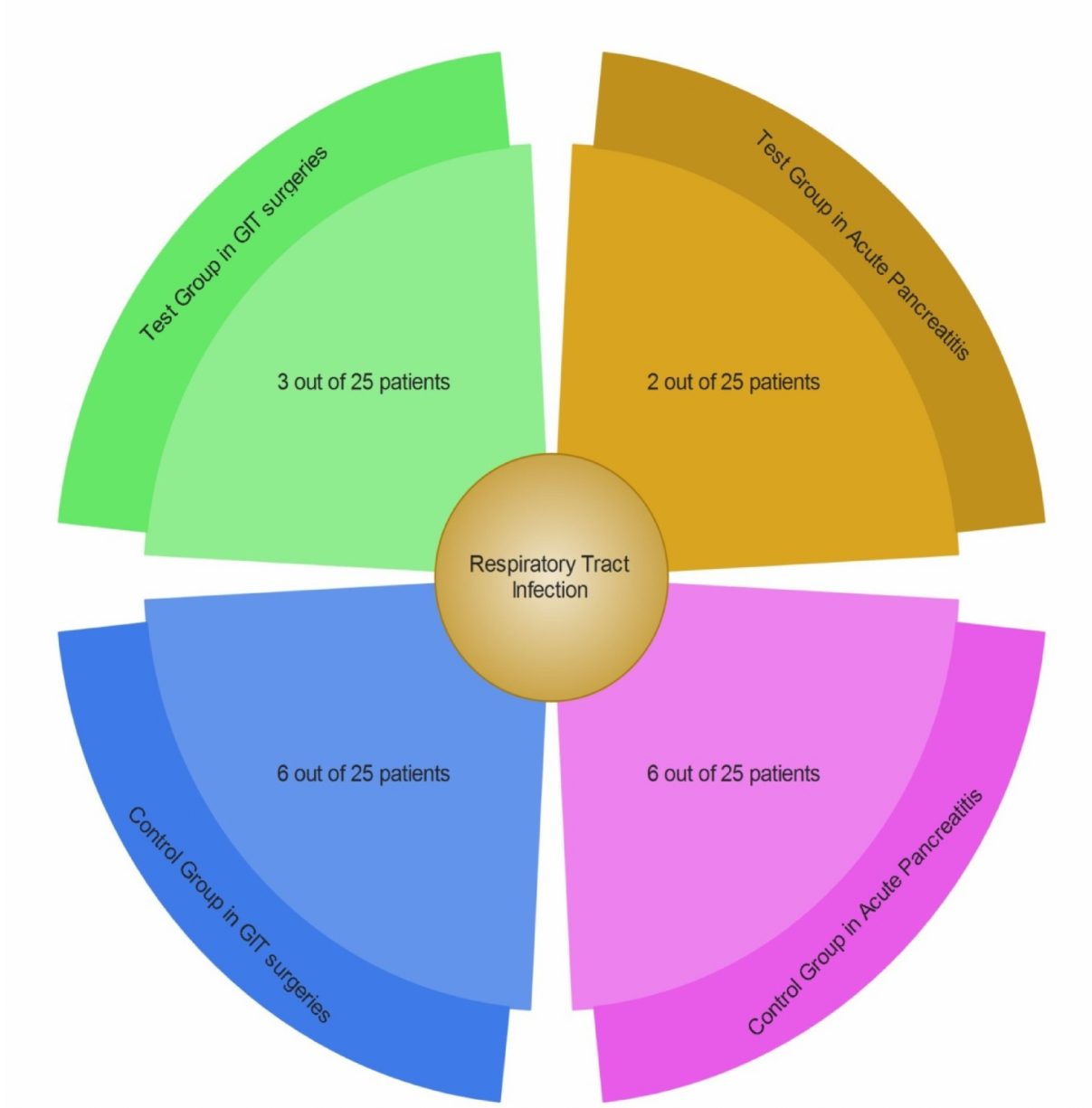
Anastomotic Dehiscence/ Peritonitis



	Test Group	Control Group
Patients with No Peritonitis	25	24
Peritonitis/ Anastomotic Dehiscence	0	1

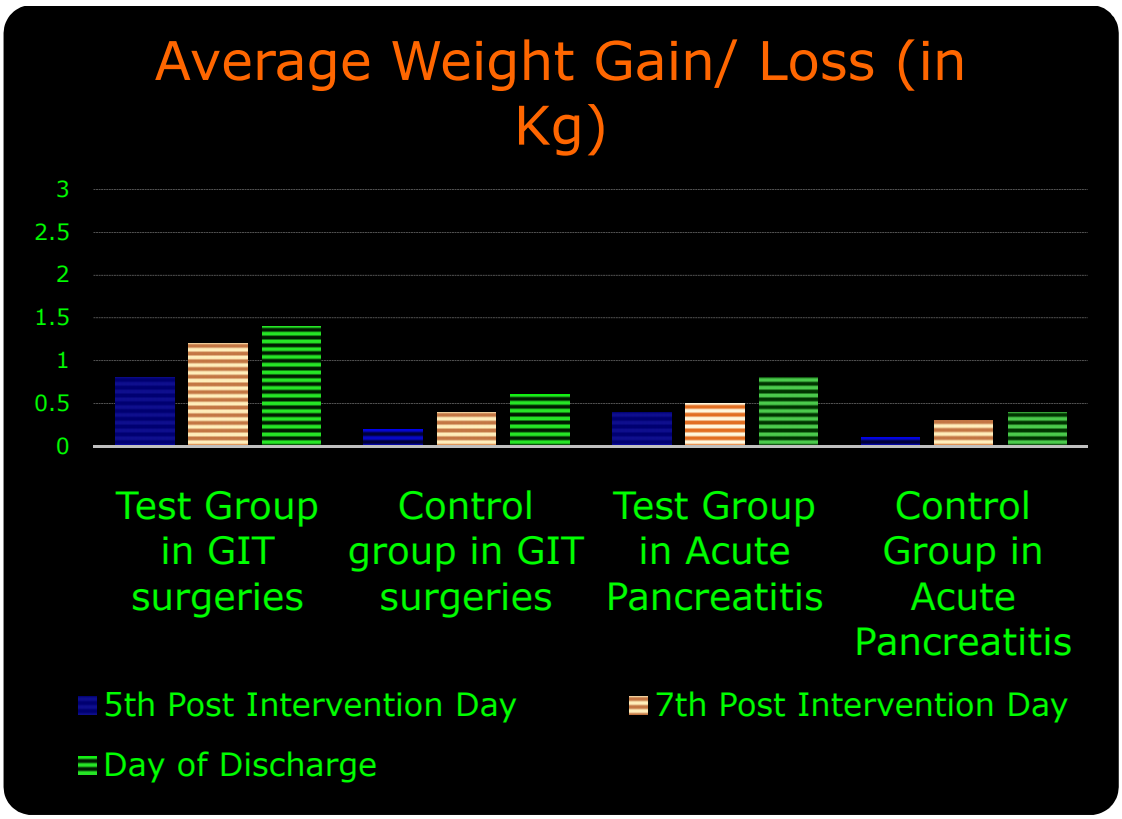
Overall, there was only one case of anastomotic dehiscence in the control group, with no such cases in the test group. It did not reach statistical significance (p = 0.09).

Respiratory Tract Infection / Aspiration



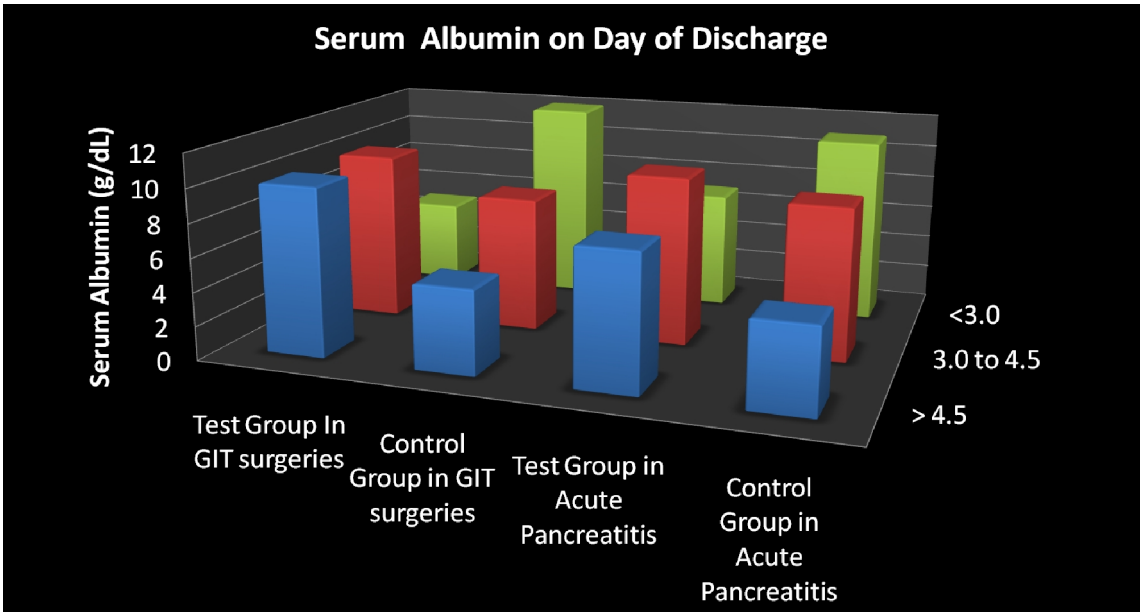
The incidence of respiratory tract infection was comparable in both the pair of groups, with a slight increase in rate in the test group in GIT surgeries. But it did not reach statistical significance (p =0.07).

Average weight gain/loss



The patients were weighed regularly from the time of admission to the day of discharge. The average gain in weight on the 5th POD was 0.8 kg in the test group, while it was 0.2 kg in the control group. In the patients with acute pancreatitis, the average gain in weight was 0.4kg and 0.1kg in the test and control groups respectively. All the values were statistically significant.

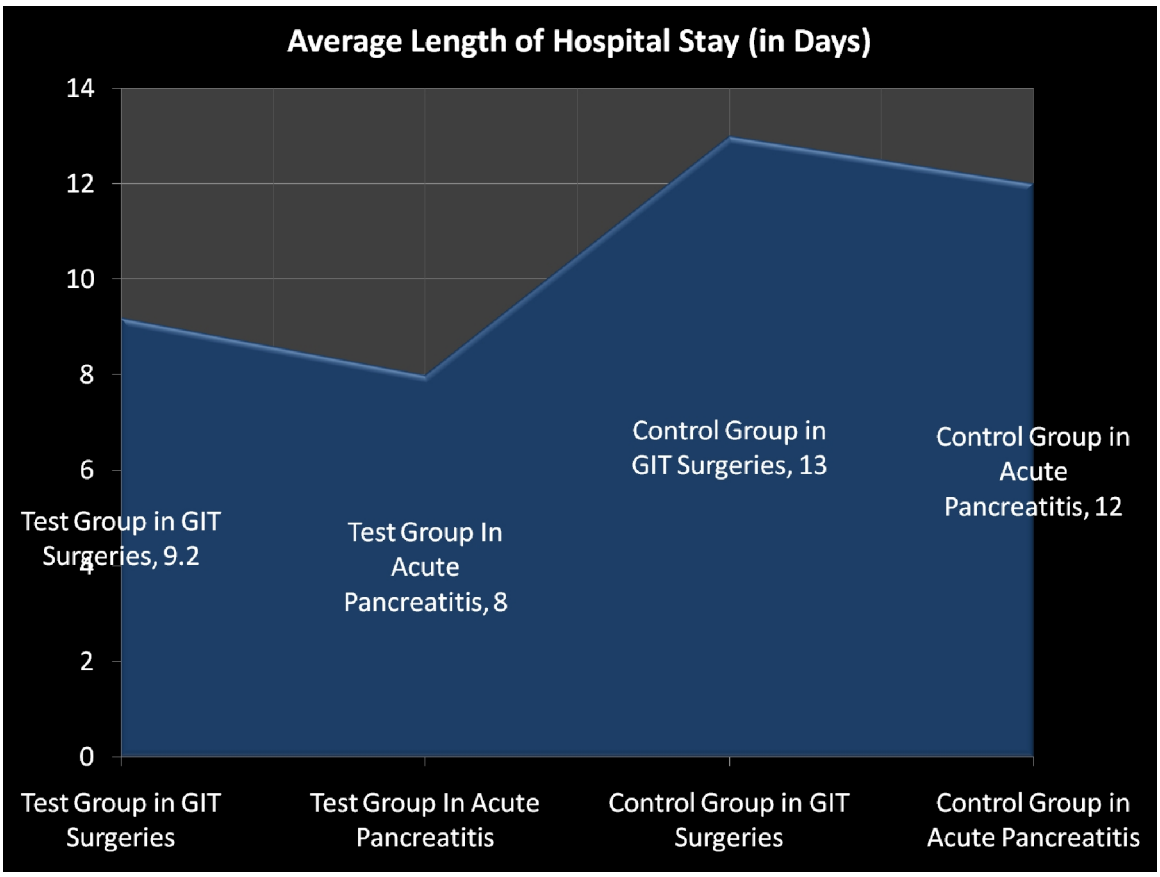
Serum Albumin



	Test Group In GIT surgeries	Control Group in GIT surgeries	Test Group in Acute Pancreatitis	Control Group in Acute Pancreatitis
> 4.5	10	5	8	5
3.0 to 4.5	10	8	10	9
<3.0	5	12	7	11

Serum albumin is a useful indicator of acute changes. In fact, the 30 day risk of mortality is often gauged by the serum albumin level. In the study, serum albumin was measured at the time of admission, on the 5th post-intervention day and at the time of discharge. The test group patients in both GIT surgeries and acute pancreatitis and higher serum albumin levels on the day of discharge when compared with control group. The graph and the data in the above table substantiate it. The values were statistically significant.

Length of Stay (LOS)



The average length of stay in the test group in GIT surgeries was 9.2 days while in its control group, it was 13 days. (p =0.03). The average LOS in patients with acute pancreatitis was 8 days in the test group and 12 days in its control group (p = 0.02).

DISCUSSION

For a long time, the functional status of the gastrointestinal tract was assessed in the surgical wards by the onset of bowel movements. The traditional teaching was "don't flog the tired horse"; comparing the adynamic bowel to an overworked stressed horse. As long as paralytic ileus persisted and the patient had not passed flatus, it was considered ideal to keep him nil by mouth. But this concept, like several others in the management of patients with acute pancreatitis and those in the post-operative period, is more empirical than evidence based. An example on similar lines would be the routine insertion of nasogastric tube preoperatively with a notion of decompressing the bowel; but many studies have shown that it does not lead to better recovery and in fact increases the chances of aspiration in a mentally obtunded patient who is recovering from anaesthesia.

Causality Dilemma of Paralytic ileus:

The traditional method of initiation of enteral nutrition was to begin when the bowel movements have started or the patient had passed flatus.Patients were maintained on dextrose-containing IV fluids and kept NPO for up to 7 days until evidence of bowel function returned. But collective data suggests that the presence of bowel sounds and the passage of flatus or stool are not absolute prerequisites for initiation of enteral nutrition. In fact in this study the mean return of bowel sounds in the test group undergoing GIT surgeries was 2.32 days (Control group – 3.4 days) while among the patients with acute pancreatitis it was 1.8 days.(Control group – 2.9

days). Both achieved statistical significance. This is brings us to a causality dilemma – "which situation leads on to the other? Should enteral feeding be delayed until the bowel starts functioning or does early feeding cause the bowel to resume its function normally?" Clearly, the results in the study show that early enteral feeding does at some level hasten the normal bowel function.

A point that must be stressed at this juncture is that an ileus must be distinguished from more ominous conditions, such as an obstruction. A prolonged ileus may be the result of intra-abdominal pathology.

Western literature is replete with studies that show that healthy patients without malnutrition undergoing uncomplicated surgery can tolerate 10 days of partial starvation (i.e., maintenance intravenous fluids only) before any clinically significant protein catabolism occurs. But in a public health system in a developing nation like ours, malnutrition is the norm. Patients are more often than not are undernourished and present in a late stage of the disease process. Earlier nutritional intervention is likely indicated in these patients with poorer preoperative/pre-interventional nutritional reserves. The attempt here is to decrease the amount of catabolism and protein breakdown, something that cannot be certainly don't with delayed initiation of feeding. The basic feature is that with enteral feeding the liver gets the first pass at the nutrients and thus promotes appropriate and economic processing of proteins.(fischer)

Protecting the anastomosis the 'wrong way'

A very frequent argument for delayed initiation of enteral feeding is that a newly constructed anastomosis must be rested before food passes through it. But it

must be reiterated that the gut secretes and reabsorbs approximately 7L fluid irrespective of oral intake; so "protecting the anastomosis" is based on a false premise. The anastomosis remains secure and is not put to any increased risk of leakage with early enteral feeding. This is aptly made out in the study where not a single case of anastomotic dehiscence occurred in the test group. Moreover, when EN is compared with parenteral nutrition, it has additional benefits such as

1. preventing gastro-intestinal mucosal atrophy,
2. attenuating the injury stress response,
3. preserving the normal gut flora, and
4. preventing microbial translocation from the gut to the blood stream by its specific trophic effects.

Furthermore studies have delineated that the prompt administration of nutrition enterally promotes the restoration of GI mucosa integrity in malnourished patients; in stark contrast to this is parenteral nutrition where such a benefit is not observed. This is because with TPN the GI mucosa continues to be permeable, in spite of the nutritional status improving. As opposed to the prevalent notion, early enteral feeding is both well tolerated and decreases the rate of post-intervention complications significantly. It minimizes the risk of undernutrition and can nullify the hypermetabolic response seen after surgery. Hence the consensus now is that in malnourished patients in the surgical wards, enteral feeding is ideal if they have a functioning GI tract.

Shortening the Convalescent Period

The shorter the recovery period of the patient in the hospital, the better it is. This was definitely the case in this study were length of stay (LOS) in the hospital was comparatively less among the test group. Moreover the days to return to normal diet was also less among the test groups. These patients also had a greater weight gain and lesser post interventional fatigue when compared with the control group. All this equates in to a shorter convalescent period and a healthier patient on the day of discharge.

Cost Effectiveness & Cost Benefit

Since parenteral feeds were not included in the study, the actual cost effectiveness could not be compared. Nevertheless, the average cost of the enteral feeds per day was around 65 rupees. This is in stark contrast to parenteral formulas, which cost around 2000 rupees per feed.

When the decreased length of stay, shorter convalescent period and the lesser post-interventional fatigue were taken in to account, early enteral feeding has a definite cost benefit.

So Why did we delay feeding to begin with?

If it is so logical to feed early using the enteral route, then why we as surgeons delay using the gut for feeding?

It can partly be attributed to the overcautious nature of the practitioners in an effort to leverage certain known and unknown factors that could jeopardize the early recovery of the patient. This over cautiousness is not entirely misplaced. Once the abdomen is closed or the ports are removed, the surgeon becomes "blind" again so to speak! If given the opportunity, he wouldn't mind strapping on an ultrasound probe to the patient and find out the most infinitesimal changes in the homeostasis.

But that is not practical, feasible, nor warranted. Over cautiousness or over indulgence on investigations is not an ideal substitute for a sound knowledge, surgical techniques and observation.

CONCLUSION

Early enteral feeding was beneficial, associated with fewer complications, and was cost effective in the study.

Nutrition is now regarded as a medical intervention, and this was aptly personified by Thoma Edison - ***The doctor of the future will no longer treat the human frame with drugs, but rather will cure and prevent disease with nutrition.***

If the gut works, use it. This is the theory behind early enteral feeding.

FUTURE RECOMMENDATIONS

Certain core investigations that would throw a better light on the effect of early enteral feeding could not be done because of their non-availability. For instance, serum pre-albumin is a better indicator for acute changes in the nutritional status of the surgical patients. Similarly, serum transferrin is quite useful in assessing the patients. Twenty four hour nitrogen balance can be calculated only if urine urea nitrogen concentration can be measured.

Formula feeds are nearly non-existent in our hospital. Only blended modular diets were used in these studies, which were made from easily available food materials. The formal introduction of commercially available formula feeds in to our hospital system could help us better control the nutritional requirements of the patient.

Food for thought – Feed the food early

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MASTER CHARTS

Master Chart for Test Group in GIT Surgeries

S. No.	IP No.	Age/Sex	Length of Stay	Diagnosis	Surgery	Day of Initiation of EN	Route of Feed	Day to pass flatus	Day to start bowel move	Weight (Kg)			Serum Albumin(g/dL)			SSI (Y/N)	Anastomotic leak/ Peritonitis (Y/N)	Respiratory tract Infection (Y/N)	Cost benefit (Y/N)
										On Admission	On 5th day	On day of discharge	On Admission	On 5th day	On day of discharge				
1	16817	33/M	8	DU Perf	Perf Closure	1	NG/ORAL	1	1	54	.5.6	54.7	4.5	4.6	4.6	N	N	N	Y
2	16900	45/F	9	Strang.Hernia	Res/Anas	1	NG/ORAL	2	2	60	60.5	60.9	3.1	3.1	3.1	N	N	N	Y
3	17122	55/M	10	Ac,Int.Obst	Res/Anas	1	NG/ORAL	2	2	58	58.4	58.8	2.7	2.8	2.8	N	N	N	Y
4	17347	34/M	8	DU Perf	Perf Closure	1	NG/ORAL	1	1	70	70.4	70.9	3.1	3.2	3.2	N	N	N	Y
5	17534	56/F	8	Gastric Perf	Perf Closure	1	NG/ORAL	2	2	68	68.5	69	4.3	4.5	4.7	N	N	N	Y
6	17647	67/M	6	C.A Oesophagus	FJ	1	FJ	2	2	49	49.6	50	3.4	3.5	3.6	N	N	N	Y
7	17734	76/M	10	Ac,Int.Obst	Res/Anas	1	NG/ORAL	3	2	58	58.1	58.3	3.1	3.3	3.3	Y	N	N	Y
8	17903	54/F	8	DU Perf	Perf Closure	1	NG/ORAL	2	2	52	52.5	53.2	3.6	3.7	3.8	N	N	N	Y
9	18345	45/M	9	Strang.Hernia	Res/Anas	1	NG/ORAL	3	3	49	49.5	50	4.4	4.6	4.5	Y	N	N	Y
10	18645	34/M	9	DU Perf	Perf Closure	1	NG/ORAL	2	2	64	64.3	64.7	4.6	4.7	4.7	N	N	Y	Y
11	18974	37/F	13	C.A Pancreas	Whipples	1	NG/ORAL	4	4	55	55.6	55.9	3.5	3.7	3.7	N	N	N	Y
12	19345	49/M	10	Ac,Int.Obst	Res/Anas	1	NG/ORAL	3	2	60	60.4	60.5	2.9	2.9	2.9	N	N	N	Y
13	19456	55/M	10	C.A Stomach	Res/GJ	1	NG/ORAL	3	3	53	53.6	53.8	4.4	4.5	4.5	Y	N	Y	Y
14	19945	12/M	8	Strang.Hernia	Res/Anas	1	NG/ORAL	2	2	47	47.3	47.6	3.6	3.8	3.9	N	N	N	Y
15	20034	45/F	7	DU Perf	Perf Closure	1	NG/ORAL	2	3	50	50.5	50.9	3.5	3.6	3.7	N	N	N	Y
16	21345	33/M	9	Strang.Hernia	Res/Anas	1	NG/ORAL	2	3	64	64.3	64.7	4.5	4.5	4.6	N	N	N	Y
17	23497	36/M	5	C.A Oesophagus	FJ	1	FJ	3	2	50	50.4	50.8	2.8	2.9	2.9	N	N	N	Y
18	25684	28/F	8	DU Perf	Perf Closure	1	NG/ORAL	2	3	73	70.3	70.6	4.1	4.2	4.2	Y	N	N	Y
19	27435	27/M	9	Strang.Hernia	Res/Anas	1	NG/ORAL	2	2	59	59.6	60	2.7	2.8	2.8	N	N	N	Y
20	28532	33/F	9	Ac,Int.Obst	Res/Anas	1	NG/ORAL	2	2	48	48.1	48.8	4.5	4.5	4.6	Y	N	N	Y
21	29435	36/F	8	Gastric Perf	Perf Closure	1	NG/ORAL	2	3	55	55.5	56	3.7	3.7	3.8	N	N	N	Y
22	29993	47/F	8	DU Perf	Perf Closure	1	NG/ORAL	3	2	70	70.3	70.8	4.5	4.6	4.7	N	N	N	Y
23	30123	49/M	9	Strang.Hernia	Res/Anas	1	NG/ORAL	2	2	60	60.6	60.7	4.4	4.5	4.5	Y	N	Y	Y
24	31245	35/F	12	C.A Stomach	Res/GJ	1	NG/ORAL	4	4	50	50.2	50.5	2.6	2.7	2.7	N	N	N	Y
25	31945	28/M	8	DU Perf	Perf Closure	1	NG/ORAL	3	2	63	63.5	64.1	4.5	4.6	4.7	N	N	N	Y

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S. No.	IP No.	Age/Sex	Length of Stay	Diagnosis	Surgery	Day of Initiation of EN	Route of Feed	Day to pass flatus	Day to start bowel move	Weight (Kg)			Serum Albumin(g/dL)			SSI (Y/N)	Anastomotic leak/ Peritonitis (Y/N)	Respiratory tract Infection (Y/N)	Cost benefit (Y/N)
										On Admission	On 5th day	On day of discharge	On Admission	On 5th day	On day of discharge				
1	16822	35/M	13	DU Perf	Perf Closure	5	NG/ORAL	5	5	58	58.2	58.2	3.6	3.7	3.8	N	N	N	N
2	16934	65/M	14	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	65	65.2	65.1	2.8	2.8	2.9	N	N	N	N
3	17167	23/M	14	DU Perf	Perf Closure	5	NG/ORAL	5	5	59	59.1	59.1	3.2	3.2	3.3	N	N	Y	N
4	17334	39/M	15	DU Perf	Perf Closure	6	NG/ORAL	5	5	55	55.1	55.3	2.5	2.6	2.6	Y	N	N	N
5	17523	59/M	10	Gastric Perf	Perf Closure	6	NG/ORAL	5	5	65	65.2	65.2	2.7	2.7	2.8	N	N	Y	N
6	17677	65/M	9	C.A Stomach	Res/GJ	6	NG/ORAL	6	5	49	49.2	49.2	4.3	4.5	4.6	Y	N	N	N
7	17756	59/F	18	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	45	45.2	45.4	3.3	3.4	3.6	Y	Y	N	N
8	17934	45/F	10	Strang.Hernia	Res/Anas	5	NG/ORAL	5	5	67	67.2	67.2	2.8	2.9	2.9	N	N	N	N
9	18344	56/M	11	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	56	56.1	56.2	2.6	2.7	2.7	Y	N	N	N
10	18656	43/M	12	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	67	67.1	67.2	4.5	4.6	4.7	N	N	Y	N
11	18998	28/F	13	DU Perf	Perf Closure	5	NG/ORAL	5	5	73	73.1	73.1	3.8	3.8	3.9	N	N	N	N
12	19306	45/M	11	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	65	65.1	65.2	3.9	3.0	2.9	N	N	N	N
13	19445	54/F	12	C.A Stomach	Res/GJ	6	NG/ORAL	6	5	49	49.1	49.2	3.5	3.7	3.9	Y	N	Y	N
14	19923	27/M	13	Strang.Hernia	Res/Anas	5	NG/ORAL	5	5	58	58.1	58.2	4.4	4.4	4.5	N	N	N	N
15	20056	48/F	14	Ac,Int.Obst	Res/Anas	5	NG/ORAL	5	5	65	65.1	65.2	3.7	3.7	3.8	Y	N	Y	N
16	21389	37/M	9	Strang.Hernia	Res/Anas	5	NG/ORAL	5	5	63	63.2	63.1	2.5	2.5	2.6	N	N	N	N
17	23456	38/F	8	Strang.Hernia	Res/Anas	2	NG/ORAL	1	1	51	51.1	51.2	4.4	4.5	4.6	N	N	N	N
18	25623	34/F	10	DU Perf	Perf Closure	5	NG/ORAL	5	5	47	47.1	47.2	2.6	2.6	2.7	Y	N	N	N
19	27498	39/M	15	Strang.Hernia	Res/Anas	6	NG/ORAL	6	5	45	45.1	45.1	3.5	3.5	3.5	N	N	N	N
20	28563	36/F	13	DU Perf	Perf Closure	5	NG/ORAL	5	5	53	53.2	53.2	2.6	2.7	2.7	Y	N	N	N
21	29420	39/F	14	Gastric Perf	Perf Closure	5	NG/ORAL	5	5	56	56.1	56.3	3.8	4.3	4.7	N	N	N	N
22	29993	42/F	15	DU Perf	Perf Closure	5	NG/ORAL	6	5	48	48.1	48.3	3.3	3.5	3.6	N	N	N	N
23	30183	55/M	10	C.A Stomach	Res/GJ	6	NG/ORAL	6	6	63	63.1	63.1	2.8	2.9	2.9	Y	N	Y	N
24	31264	56/F	14	DU Perf	Perf Closure	5	NG/ORAL	5	5	59	59	59.2	2.7	2.8	2.8	N	N	N	N
25	31905	49/M	13	DU Perf	Perf Closure	5	NG/ORAL	5	5	61	61	61.1	3.1	3.2	3.3	N	N	N	N

Master Chart for Test Group in Acute Pancreatitis

S. No.	IP No.	Age/Sex	Length of Stay	Diagnosis	Day of Initiation of EN	Route of Feed	Day to pass flatus	Day to start bowel move	Weight (Kg)			Serum Albumin(g/dL)			Respiratory tract Infection (Y/N)	Cost benefit (Y/N)
									On Admission	On 5th day	On day of discharge	On Admission	On 5th day	On day of discharge		
1	16634	35/M	8	Acute Pancreatitis	1	NG/ORAL	1	1	55	55	55.1	4.2	4.2	4.6	N	N
2	17003	43/M	9	Acute Pancreatitis	1	NG/ORAL	1	1	53	53.1	53.1	3.	3.7	3.8	N	N
3	17134	45/M	8	Acute Pancreatitis	1	NG/ORAL	5	1	65	65	65.1	3.3	3.3	3.6	N	N
4	17345	39/F	8	Acute Pancreatitis	1	NG/ORAL	2	2	58	57.8	57.8	3.4	3.4	3.5	N	N
5	17508	48/M	8	Acute Pancreatitis	1	NG/ORAL	1	1	60	60.1	60.1	4.5	4.5	4.5	Y	N
6	17623	33/M	8	Acute Pancreatitis	1	NG/ORAL	6	5	55	55.1	55.1	3.1	3.1	3.4	N	N
7	17776	45/M	8	Acute Pancreatitis	1	NG/ORAL	5	5	49	49.1	49.1	3.0	3.0	2.9	N	N
8	17934	45/M	9	Acute Pancreatitis	1	NG/ORAL	2	2	67	67.1	67.1	2.6	2.6	2.8	N	N
9	18356	34/M	7	Acute Pancreatitis	1	NG/ORAL	1	1	56	56.1	56.1	4.5	4.5	4.7	N	N
10	18667	47/M	8	Acute Pancreatitis	1	NG/ORAL	5	5	67	67.1	67.1	3.3	3.3	3.6	Y	N
11	18946	29/M	8	Acute Pancreatitis	1	NG/ORAL	5	5	68	67.9	68	3.0	3.0	2.7	N	N
12	19378	49/M	8	Acute Pancreatitis	1	NG/ORAL	1	1	65	65.1	65.1	4.3	4.3	4.6	N	N
13	19449	45/M	7	Acute Pancreatitis	1	NG/ORAL	2	2	49	49.1	49.1	2.9	2.9	2.9	N	N
14	19934	28/M	9	Acute Pancreatitis	1	NG/ORAL	3	3	58	58.1	58.1	3.3	3.3	3.5	N	N
15	20024	45/M	8	Acute Pancreatitis	1	NG/ORAL	1	1	65	65.1	65	4.1	4.3	4.5	N	N
16	21389	34/M	8	Acute Pancreatitis	1	NG/ORAL	2	2	63	63	63	2.6	2.7	2.8	N	N
17	23434	37/M	10	Acute Pancreatitis	1	NG/ORAL	1	1	51	51.1	51	3.5	3.6	3.9	N	N
18	25645	33/M	8	Acute Pancreatitis	1	NG/ORAL	1	1	47	47.1	47.1	2.6	2.6	2.6	N	N
19	27434	34/M	7	Acute Pancreatitis	1	NG/ORAL	6	5	45	45.1	45.1	3.3	3.4	3.6	N	N
20	28567	36/M	8	Acute Pancreatitis	1	NG/ORAL	3	3	53	53.2	53.2	3.3	3.3	3.4	N	N
21	29478	33/F	6	Acute Pancreatitis	1	NG/ORAL	4	4	56	56.1	56	4.1	4.2	4.5	N	N
22	29989	48/M	8	Acute Pancreatitis	1	NG/ORAL	6	5	48	48.1	48	3.3	3.4	3.5	N	N
23	30145	50/M	8	Acute Pancreatitis	1	NG/ORAL	4	4	63	63.1	63	2.5	2.6	2.7	N	N
24	31245	44/M	6	Acute Pancreatitis	1	NG/ORAL	5	5	59	59	59.2	3.6	3/7	3.9	N	N
25	31945	45/M	7	Acute Pancreatitis	1	NG/ORAL	4	4	61	61	61	4.4	4.5	4.6	N	N

Master Chart for Control Group in Acute Pancreatitis

S. No.	IP No.	Age/Sex	Length of Stay	Diagnosis	Day of Initiation of EN	Route of Feed	Day to pass flatus	Day to start bowel move	Weight (Kg)			Serum Albumin(g/dL)			Respiratory tract Infection (Y/N)	Cost benefit (Y/N)
									On Admission	On 5th day	On day of discharge	On Admission	On 5th day	On day of discharge		
1	16675	35/M	10	Acute Pancreatitis	5	NG/ORAL	5	5	55	55	55.1	3.3	3.5	3.7	N	N
2	16967	43/M	11	Acute Pancreatitis	5	NG/ORAL	5	5	53	53.1	53.1	2.6	2.7	2.8	N	N
3	17156	23/M	11	Acute Pancreatitis	5	NG/ORAL	5	5	65	65	65.1	3.5	3.5	3.6	Y	N
4	17325	39/M	9	Acute Pancreatitis	2	NG/ORAL	1	1	58	57.8	57.8	4.2	4.5	4.6	N	N
5	17506	48/M	10	Acute Pancreatitis	3	NG/ORAL	5	5	60	60.1	60.1	3.3	3.4	3.5	Y	N
6	17649	33/M	9	Acute Pancreatitis	6	NG/ORAL	6	5	55	55.1	55.1	2.8	2.8	2.9	N	N
7	17797	37/M	18	Acute Pancreatitis	5	NG/ORAL	5	5	49	49.1	49.1	3.3	3.4	3.7	N	N
8	17909	27/M	10	Acute Pancreatitis	5	NG/ORAL	5	5	67	67.1	67.1	2.7	2.7	2.8	N	N
9	18346	46/M	9	Acute Pancreatitis	5	NG/ORAL	5	5	56	56.1	56.1	4.5	4.6	4.7	N	N
10	18695	43/M	10	Acute Pancreatitis	2	NG/ORAL	1	1	67	67.1	67.1	3.0	3.1	3.3	Y	N
11	18956	28/M	10	Acute Pancreatitis	5	NG/ORAL	5	5	68	67.9	68	2.6	2.6	2.7	N	N
12	19394	45/M	12	Acute Pancreatitis	5	NG/ORAL	5	5	65	65.1	65.1	3.1	3.2	3.2	N	N
13	19406	47/M	11	Acute Pancreatitis	6	NG/ORAL	6	5	49	49.1	49.1	4.5	4.5	4.8	Y	N
14	19923	27/M	13	Acute Pancreatitis	5	NG/ORAL	5	5	58	58.1	58.1	2.6	2.6	2.7	N	N
15	20096	48/M	14	Acute Pancreatitis	5	NG/ORAL	2	1	65	65.1	65	3.3	3.4	3.4	Y	N
16	21336	37/M	12	Acute Pancreatitis	5	NG/ORAL	5	5	63	63	63	4.4	4.5	4.5	N	N
17	23407	38/M	13	Acute Pancreatitis	3	NG/ORAL	2	2	51	51.1	51	2.9	2.9	2.9	N	N
18	25627	34/M	9	Acute Pancreatitis	5	NG/ORAL	5	5	47	47.1	47.1	3.5	3.6	3.6	N	N
19	27456	39/M	13	Acute Pancreatitis	6	NG/ORAL	6	5	45	45.1	45.1	4.2	4.3	4.6	N	N
20	28586	36/M	114	Acute Pancreatitis	5	NG/ORAL	5	5	53	53.2	53.2	3.0	3.0	2.9	N	N
21	29490	39/F	10	Acute Pancreatitis	3	NG/ORAL	2	2	56	56.1	56	2.6	2.7	2.8	N	N
22	29945	42/M	113	Acute Pancreatitis	5	NG/ORAL	6	5	48	48.1	48	3.3	3.5	3.6	N	N
23	30103	55/M	10	Acute Pancreatitis	6	NG/ORAL	6	6	63	63.1	63	2.6	2.7	2.9	Y	N
24	31229	45/M	13	Acute Pancreatitis	5	NG/ORAL	5	5	59	59	59.2	3.7	3.7	3.7	N	N
25	31969	49/M	13	Acute Pancreatitis	5	NG/ORAL	5	5	61	61	61	3.5	3.5	3.5	N	N